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CHEST PAIN IN PATIENTS WITH MITRAL STENOSIS, WITH PARTICULAR REFERENCE TO SO-CALLED "HYPERCYANOTIC ANGINA"*

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PATIENTS with mitral stenosis often complain of chest pain. Pain also frequently occurs from causes unrelated to the heart, but such pain is not discussed in this paper. There are four main varieties of chest pain especially associated with mitral stenosis: that of cardiac neurosis; that of active rheumatic carditis; angina pectoris; and so-called "hypercyanotic angina." In none is the pain directly produced by the stenosed mitral valve. In addition, patients with mitral stenosis are prone to develop pulmonary infarction and may have repeated episodes. Pain may be present either as pleural pain of an associated pleuritis or as substernal distress, especially with massive pulmonary infarctions or emboli.

PAIN OF CARDIAC NEUROSIS

Without doubt, the type of chest pain that patients with mitral stenosis most commonly complain of is that of cardiac neurosis; such pain, for which no organic cause can be demonstrated, is largely psychogenic in origin and usually dependent on an anxiety state. It is very variable, ranging from aching soreness or pins-and-needles sensation, to sharp, stabbing or lancinating, and is usually felt in the apical region, occasionally all over the precordium and even sometimes in the chest and down the arms. It has no constant relation to exertion and frequently persists for hours or days. The pain is often attended by precordial hyperesthesia. Breathlessness, which in reality consists in sighing or a sense of inability to get enough air into the lungs, is a common complaint. There may or may not be associated manifestations of neurocirculatory asthenia such as tachycardia, palpitation, cyanotic cold extremities, fatigability and poor response to effort. Part of the usual diagnosis of such functional pain is the absence of evidence of organic heart disease, but not uncommonly patients with unquestionable organic heart disease, including mitral stenosis, have unmistakable functional pain. The association is obviously explained by natural anxiety when patients know of their heart disease and its dangers. Does marked enlargement of the heart, such as some patients with mitral stenosis have, in itself cause precordial pain? Certainly, patients with very large hearts beating forcibly against the chest wall are likelier to have heart consciousness than normal persons, but such a sensation rarely, if ever, reaches the degree of true pain in the absence of a neurotic or anxiety factor. The following is a typical example of a patient with the functional type of pain.

CASE 1.§ The patient, a 42-year-old woman, who had rheumatic fever in her youth, but following this led a normal life, was married and had two children. Following the birth of the second child, "she did not regain her strength," but suffered from breathlessness at her accustomed tasks and complained of a great sense of fatigue and of sleeplessness and precordial pain. She was seen in consultation at this time by an able cardiologist, who found mitral stenosis and apparently interpreted this as the cause of her complaints and stressed merely the necessity of a cautious regime on her and her husband. For 20 years, the patient was up only a few hours a day, rarely went out, and was almost constantly conscious of her heart. Distressing precordial pain was the most prominent of her many symptoms. She never developed congestive failure. Suddenly, after 20 years of cardiac neurosis and no heart failure, without warning, she developed a right hemiplegia, probably from an embolus. She recovered partially from the paralysis, but her personality clearly changed. She became cheerful, fearless and free from complaints. There

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§This case has previously been cited by Ellis and Hamilton.[1]

was no more mention of consciousness of the heart and heart pain. The patient struggled about the house doing more work than she had done at any time for the previous 20 years, and remained up all day. This state continued until she was lost sight of several years later.

The pain in this patient was of the severe type described above. For twenty years, she was disabled by a functional state, aggravated undoubtedly by the misinterpretation of symptoms by the cardiologist who first saw her. The spontaneous relief of the neurotic symptoms with the onset of an organic disability, the hemiplegia, is of interest and has been discussed elsewhere.[1]

Pain of Active Rheumatic Carditis

It is well known that pain in the chest may accompany active rheumatic carditis. This symptom has been thoroughly discussed by Swift and Hitchcock.[2] This type of pain is said to be commonest in acute recurrences of rheumatic fever, and accordingly should sometimes appear in patients with mitral stenosis. The following case illustrates most of the typical features of this type of pain.

Case 2. A. M., a 37-year-old woman, had had acute rheumatic fever at the age of 14, and had been known to have mitral stenosis and auricular fibrillation for several years. She entered the Boston City Hospital because of persistent, dull, heavy, aching pain, located in the midsternal region and nonradiating. In association with this was hyperesthesia of the precordial region of sufficient degree to make cardiac percussion uncomfortable to the patient. She had, on admission, in addition to the physical signs of her valvular lesion, mild migratory joint pains, a slight fever, leukocytosis and elevated sedimentation rate. As these latter findings disappeared so did the patient's precordial pain and hyperesthesia, and she recovered uneventfully.

The pain in this case was consistent with cardiac neurosis, but there were no other neurotic symptoms, and the occurrence and subsidence of the pain in conjunction with the onset and disappearance of signs of rheumatic infection indicate its relation to active carditis. Active rheumatic carditis may be accompanied by pericarditis, and if the diaphragmatic or a certain part of the pleural pericardium is involved, there may be pain in the precordium, the root of the neck or upper abdomen.

Angina Pectoris

It has long been recognized that angina pectoris is rarely associated with mitral stenosis. White[3] found mitral stenosis in but 2 of 500 cases of angina. Hamilton and Thomson[4] found but one patient with an unmistakable story of effort limited by angina pectoris among many hundred young women of child-bearing age with mitral stenosis. Although evidence exists that rheumatic fever may produce or hasten organic disease of the coronary arteries,[5] the rarity of angina pectoris except in patients in the older age group and usually with other clinical evidence of arteriosclerosis makes it probable that when this symptom does occur it is usually due to a superimposed coronary atherosclerosis and does not differ essentially from angina pectoris occurring in patients without valvular heart disease. Blackford[6] has recently reported 2 cases of angina in patients with mitral stenosis who had normal coronary arteries. One of these cases presented features suggesting that it falls into the group of "hypercyanotic angina," discussed below. Uncommon as angina pectoris is in association with mitral stenosis, acute myocardial infarction is even rarer. White[7] did not encounter a single case of mitral stenosis in a series of 200 cases of myocardial infarction. The characteristics of the pain of angina pectoris are too well known to need description. The following is an example of a case of angina pectoris and mitral stenosis.

Case 3. P. R., a 50-year-old man, entered the hospital in acute congestive failure and stated that for the previous 5 weeks he had had increasingly frequent attacks of severe precordial pain and tightness, precipitated by even mild exertion and radiating down the left arm in the form of "numbness" or a "twisting" pain. The patient had a clear history of several attacks of rheumatic fever in childhood and typical physical and x-ray findings of mitral stenosis. The patient's failure responded to the usual treatment, but when allowed up and about he had frequent return of his precordial pain on effort.

Hypercyanotic Angina

Perhaps the most dramatic precordial pain occurring in patients with mitral stenosis is the unusual type of paroxysmal precordial or substernal pain, called "hypercyanotic angina" by some authors. This pain, since it occurs principally in cases of severe mitral stenosis, may well bear some direct relation to the valvular lesion. The pain is severe and may be excruciating, lasting hours or even days in extreme cases. It is clinically indistinguishable from the pain of an acute myocardial infarction but is associated with none of the electrocardiographic changes, fever and leukocytosis that accompany the latter, and at post-mortem examination there is no evidence of infarction. The following cases are presented as extreme examples of this type.

Case 4. E. R., a 33-year-old woman, gave a history of rheumatic fever in young adult life, and signs of progressively diminished cardiac reserve for 6 years. For 2½ years, she had suffered increasingly frequent and severe attacks of dyspnea, orthopnea, cyanosis and very severe precordial pain, burning in character and radiating to the left side and back of the chest, with a sensation of warmth in the left arm. The first attack was of 5 days' duration and was repeated in varying degrees of severity during

the months prior to entry. Between attacks, the patient was quite well except for mild exertional dyspnea and the occurrence of five hemoptyses.

On entry, the patient seemed acutely ill, complaining of severe pain, with marked cyanosis, moderate orthopnea and some venous distention. There was slight congestion of the right lung base. The heart was enlarged, with signs of mitral stenosis, and the liver was enlarged and slightly tender. The blood pressure was 68/30, and rose to a normal level following the attack. The pain responded to oxygen and sedation, and the patient was then well except for low-grade fever and leukocytosis. Attacks of pain recurred several times during the hospital stay, always with severe cyanosis, pulmonary edema and collapse, and oxygen seemed always to be the deciding factor in their relief. Electrocardiograms showed T-wave abnormalities reflecting digitalis therapy, and they showed no change during the entire course except for a shift from sinus rhythm to auricular fibrillation. The patient died during the 12th hospital week in another severe attack of precordial pain.

Post-mortem examination revealed a markedly stenosed, thickened and fixed "button-hole" mitral valve, a normal set of coronary arteries, which were patent and showed no sclerosis, and a myocardium that was hypertrophic in the region of the right ventricle but otherwise unremarkable. The pulmonary changes in this case were very marked and have been reported in detail by Parker and Weiss[8] in their study of pulmonary vascular changes secondary to mitral stenosis. These changes consisted in general of marked capillary dilatation, with a tendency to aneurysmal bulging into the alveolar lumens, thickening of the capillary basement membrane, a tendency to interstitial fibrosis in the alveolar walls and in many areas the appearance of a cuboidal type of epithelium in the alveoli. The larger arterial vessels showed a proliferative endarteritis.

Case 5. J. P., a 65-year-old woman who had had acute rheumatic fever in childhood, entered the hospital because of several weeks of intermittent severe "pressure-pain" in the left chest, with radiation to the left arm, which had become almost constant for the 2 weeks prior to admission and was unrelated to activity. On entry, the patient showed orthopnea, cyanosis and extreme prostration. The heart was totally irregular, and there was a rough apical systolic murmur. There were slight pulmonary congestion and minimal peripheral edema. The blood pressure was 110/50. The patient continued to be apparently in extremis throughout the entire 1st hospital week, after which she improved gradually but had frequent recurrences of pain, and she was discharged after 4 months.

During the succeeding year, the patient entered three more times for similar severe episodes, the last admission following 5 weeks of apparently continuous pain and cyanosis. At all these admissions, there was slight objective evidence of pulmonary congestion and none of peripheral edema. At the last entry, physical and electrocardiographic findings were still those of the first admission. The patient died on the 32nd hospital day following another severe attack.

Autopsy showed a 300-gm. heart, the coronary vessels showing normal patency but mild atheroma, and the myocardium showed only microscopic perivascular fibrosis. The mitral valve was thickened and stenosed, and the pulmonary vessels showed changes similar to those in Case 4 although somewhat less marked.

Case 6. M. Y. entered the hospital six times between the ages of 48 and 58. She gave a history of two attacks of acute rheumatic fever in early adult life, and for 18 months prior to entry she had had bouts of severe precordial pain radiating down the left arm and accompanied by cyanosis and dyspnea. On each entry, the story was striking, with severe and prolonged precordial pain and cyanosis — lasting several hours at a time and not related to exertion. There was evidence of slight persistent pulmonary congestion, but no peripheral edema, throughout each entry. Attacks recurred at frequent intervals, and were best relieved on those occasions when the patient received oxygen therapy in the hospital. During one attack, the blood pressure rose from 150/100 to 200/130, returning subsequently to its previous level; in another attack, the blood pressure fell. The patient had chronic auricular fibrillation throughout her course, and marked signs of mitral valvular disease. The final hospital admission followed a month of continuous pain and congestive heart failure and ended in death. The only electrocardiographic change from the findings at the first admission until death was a gradual shift of the axis toward the right. Unfortunately, permission for post-mortem examination was denied.

Discussion

In brief, the picture presented by the last 3 cases and by a few similar ones in the literature is as follows. The pain is severe, squeezing or grinding, located in the precordium or beneath the sternum, radiating to the left shoulder and arm, bearing no clear relation to exertion, emotion or stress, and occurring in paroxysms that may last for hours or days. In addition, there are usually dyspnea, orthopnea and a severe degree of cyanosis during the attacks, and in some cases marked circulatory collapse. Peripheral venous congestion may or may not be present. Although our patients all had some congestion of the lungs at the time of the attacks, the relation of the pain to heart failure is by no means constant. The picture that these patients present is often unlike the usual manifestations of heart failure. Their cyanosis may be extreme, and their dyspnea and orthopnea well marked at a time when the signs of pulmonary congestion are surprisingly few. Roentgenograms of the lung fields are likelier to show markedly increased hilar markings and shadows of enlarged pulmonary vessels fanning out from the hilum than the usual basal congestion.

There is not the fever, leukocytosis or electrocardiographic pattern of an acute myocardial infarction, and the changes in blood pressure are irregular and in no way characteristic. Oxygen relieves the pain much more effectively than vasodilator drugs. Post-mortem examination shows a high degree of mitral stenosis, with secondary myocardial hypertrophy and pulmonary vascular changes.

This symptom complex, which is by no means new, is seen occasionally in most large clinics. It is, however, confusing and often leads, as in all 3 of our cases, to a mistaken diagnosis of myocardial

infarction. Striking as it is, it has received relatively little attention in the medical literature and has escaped mention in most of the standard texts. It is probable that mild grades of this syndrome occurring in patients with marked mitral stenosis are not so uncommon as observers have thought.

The first definite mention of chest pain as a feature of mitral stenosis is contained in an article by Nothnagel,[9] who, in 1891, presented statistics concerning the frequency of occurrence of pain in all types of valvular heart disease. The aortic lesions, of course, led the field, but it is of interest that he recorded pain in 18 per cent of his cases of mitral stenosis. He further reported a case of severe prolonged precordial pain in a thirty-six-year-old woman whose story is very similar to that in Cases 4, 5 and 6.

Since the turn of the century, there have been a few scattered articles dealing with pain of this type, further describing and sometimes attempting to explain it. Vaquez and Giroux,[10] in 1908, described the clinical picture of *angina pulmonaire hypercyanotique*, which they considered a consequence of pulmonary vascular abnormalities. Posselt,[11] in the same year, mentioned *Dyspragia intermittens angiosclerotica pulmonalis*, apparently the same syndrome, as one of the criteria to be used for the clinical diagnosis of pulmonary arteriosclerosis, and suggested that the pain might be produced by distention of the pulmonary arteries caused by pulmonary arteriolar spasm. In 1911, Frugoni[12] contrasted the *dolor pallidus* of ordinary effort angina with the *dolor caeruleus* in patients with sclerosis of the pulmonary vascular system. He believed that the pain had a distinctive type of radiation, differing from that of ordinary angina, from the precordium deep into the chest, an opinion that was echoed by Engelen[13] in 1923. Arrillaga,[14] in discussing Ayerza's syndrome, comments on the occasional occurrence of pain of the type described by Posselt.

The writings of Brenner[15] on the pathology of the pulmonary circulation contain some further mention of hypercyanotic angina occurring in patients with secondary pulmonary arteriosclerosis. Of particular interest, however, is the case that he cited of an eleven-year-old boy who had primary pulmonary arteriosclerosis of sufficient degree to cause marked right ventricular hypertrophy. The description of the pain is identical with that in our cases. This is, interestingly enough, the only case of this phenomenon in the literature occurring in a male. Brenner suggests that the pain is the result of myocardial anoxia, which is caused by the severe anoxemia.

Brill and Krygier,[16] in a recent review of primary pulmonary vascular sclerosis, commented on the occurrence of chest pain in 4 of the 20 cases they assembled from the literature, including 1 reported by themselves, and considered that the pain was probably due to acute coronary insufficiency consequent to a diminished volume of blood delivered to the left ventricle through an obstructed pulmonary circulation.

In the foregoing discussion, attention has been devoted primarily to disorders of the pulmonary circulation. Although in some cases these disorders are associated with mitral stenosis, they frequently occur in patients suffering from chronic pulmonary disease or even primary pulmonary vascular disease.

There has been some comment in the literature specifically of the association of pain of this type and mitral stenosis. Parker and Weiss[8] mentioned pain and cyanosis prominently among the symptoms of their cases of mitral stenosis with severe secondary pulmonary changes, and commented on their absence in the control cases with a normal pulmonary circulation. They made the suggestion that the cyanosis is due to a number of factors. The alveolar capillaries are so dilated as to allow simultaneous passage of several columns of red cells instead of the normal one, with consequent inefficient oxygenation, and the structural changes that they describe are such as to increase greatly the thickness of the membrane through which the gaseous exchange must occur.

Some authors have attributed the pain to strictly intracardiac explanations of conditions. Sternberg,[17] in reporting the autopsy findings in a twenty-six-year-old woman with mitral stenosis who had severe precordial pain for five days preceding death, noted that the coronary arteries were unusually thin and collapsible, and suggested that the terminal precordial pain was the result of compression of the left coronary artery in its course between the left auricle and the pulmonary conus, which were distended as a consequence of the mitral stenosis. Hochrein[18] later took issue with this theory, stating that in his experience pain had been independent of the degree of decompensation. He offered an alternative explanation based on the post-mortem findings in 1 of 4 cases that he reported. Using a special technic, he demonstrated that the left coronary orifice was pulled down and distorted as a result of the contraction of a scarred mitral valve and its shortened chordae tendineae. This, he suggested, had sufficiently impaired the coronary flow to explain the pain.

It seems reasonable to assume, from the character and location of the pain, that it arises from anoxia of the myocardium. The relief of pain by oxygen that occurred in our patients certainly suggests that anoxia is the cause and that general de-

saturation of the blood with oxygen is an important factor. In addition, there is probably a decrease in the amount of blood reaching the left side of the heart through the impaired pulmonary vascular bed, and hence the left ventricular output is diminished. This in turn causes impairment of coronary blood flow, which thus further enhances the myocardial anoxia. It is likely that an abnormal heart is also a necessary part of the picture in addition to an abnormal pulmonary vascular system producing anoxemia and diminished blood flow. Although disease of the coronary arteries may be present, it is unnecessary in the production of the clinical syndrome, and, in view of the cases of primary and secondary pulmonary vascular disease cited above, mitral valvular disease is also not an essential feature. The common denominator of all cases is myocardial hypertrophy of the right ventricle.

The pain in character and duration is similar to that occasionally seen in patients with impaired coronary circulation or myocardial hypertrophy, or both, who develop coronary insufficiency as the result of paroxysmal tachycardia. In our cases, tachycardia of any degree did not occur.

In severe mitral disease, several factors could promote deficiency of the myocardial oxygen supply. In the first place, the increased load of work thrown on part of the myocardium by the damaged valve creates an abnormally high need for oxygen, and hypertrophy of the overworked portion of the myocardium, which in itself constitutes an unfavorable situation from the nutritional standpoint, makes this increased need the more difficult to meet. When one adds to this the anoxemia that is present, and an impaired coronary blood flow resulting from a cardiac output diminished by the obstruction in the pulmonary circulation, as well as the mechanical narrowing of the mitral orifice, the presence of pain is not surprising.

It is to be recognized that the foregoing explanation does not answer all the questions posed by this syndrome. One such question is the lack of definite correlation between the pain and exertion. One must assume that the capacity of these patients for physical exertion was sharply limited by dyspnea before pain occurred. When they became well compensated, oxygenation was sufficient to allow for moderate effort without pain. The lack of electrocardiographic change during the attacks is of interest, since it is well known that electrocardiographic abnormalities may develop during coronary insufficiency occurring in angina pectoris or from other causes. However, such alterations in the electrocardiogram are by no means invariable in angina pectoris, and it is quite possible that further electrocardiographic studies on patients with hypercyanotic angina will reveal on occasion alterations consistent with coronary insufficiency. Furthermore, since the myocardial anoxia is general and not focal, striking electrocardiographic abnormalities are less likely to occur.

The one-sided sex distribution of the reported cases is puzzling, but may diminish or disappear as more cases come to light.

Summary and Conclusions

Four types of chest pain occurring in patients with mitral stenosis are described, and cases illustrating each type are reported. The types of pain are: that of cardiac neurosis; that of acute rheumatic carditis; angina pectoris; and so-called "hypercyanotic angina."

The literature bearing on hypercyanotic angina is discussed.

It is suggested that this type of pain is due to anoxia in a hypertrophic and overworked myocardium. This anoxia is explained as the combined result of severe organic changes in the pulmonary vessels, with pulmonary congestion and arterial anoxemia, in addition to reduced blood flow to the left ventricle and hence to the coronary arteries.

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THE SYNDROME OF CERVICAL RIB WITH SUBCLAVIAN ARTERIAL THROMBOSIS AND HEMIPLEGIA DUE TO CEREBRAL EMBOLISM*

Report of a Case

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BOSTON

IT IS not generally appreciated that thrombosis of the subclavian artery may in rare cases be the source of cerebral embolism. Symonds,[1] in 1927, reported 2 cases in which absent arterial pulsations in the right arm, apparently resulting from cervical ribs, had been complicated by the sudden development of left hemiplegia. His explanation was that ". . . pressure of the cervical rib upon the right subclavian artery led to thrombosis, that the thrombus extended backwards to the point of junction with the right common carotid, and a portion of it becoming broken off formed an embolus in one of the terminal branches of the right middle cerebral artery."

The fatal outcome in such a case was described one year later by Yates and Guest.[2] Their patient had complained of progressive pain and weakness of the right hand and forearm for some months. An absent arterial pulsation in the right upper extremity and an ununited fracture of the right clavicle were found. Before treatment could be instituted, the patient was taken suddenly ill with pain in the head and throat, loss of vision, unconsciousness, left hemiplegia progressing to quadriplegia, coma and death on the seventh day. Autopsy revealed an old ununited fracture of the right clavicle, with backward and downward displacement toward the right subclavian artery. This vessel was filled with an organized and very adherent thrombus, which extended to within 1.0 cm. of its junction with the right common carotid artery. A well-marked embolus was found at the division of the basilar artery into its terminal branches. The authors suggested that in this case the embolism had occurred through the escape of a fragment of the thrombus up the right vertebral artery.

More recently, Smith[3] has reported a similar case, in which the autopsy findings conformed even more closely to Symonds's original explanation of the course of the embolism. The patient, a twenty-two-year-old seaman, suddenly developed a left hemiplegia. He had occasionally complained of a mild, tingling sensation in the right arm and hand that had never incapacitated him. At the time of the cerebral accident, no pulse could be obtained anywhere in the right upper extremity. Shortly thereafter, the patient lapsed into coma and died. Autopsy revealed a thrombosis of the right axillary and right subclavian arteries, apparently originating in a small, sacculated congenital aneurysm attached to the right axillary artery. The thrombus extended as far as the origin of the right common carotid artery, and a large embolus was found occluding the right internal carotid artery where it enters the carotid canal.

In a brief review of the literature, only one other case could be found that may represent the combination of events first recognized by Symonds. This was reported by Gould[4] in 1884. Although the author apparently did not recognize the pathogenesis of the symptoms, calling it a case of "obliterative arteritis," his description is very clear, and the clinical course similar to the case to be reported. It may be summarized as follows:

> C. B., age 19, a laborer in a brickfield on the Isle of Wight, was admitted to the London Temperance Hospital on October 13, 1883. At the end of April, 1883, he noticed the fingers of his right hand getting "dark" in color. The hand seemed weak; when at work it quickly became cold and then there was a "sickening pain" in it.
>
> State on admission: the limb below the elbow was distinctly colder than the opposite one. Just above the elbow all pulsation ceased. The radial artery was a prominent, hard, pulseless cord. A great prominence of the right subclavian artery above the clavicle was noted; it projected under the skin as a tense pulsating cylinder which was bulging forward even beyond the level of the clavicle. Springing from each side of the lower two cervical vertebras was found a hard, immovable, irregular mass, apparently bony. When the subclavian artery was pressed against the mass in a particular direction, a thrill was produced in it.

In a second communication, three years later, the author[5] stated:

> This patient was shown to the Society two years ago. Since then he remained at work as a labourer until six months ago, when he was attacked with numbness in the right arm and forearm, accompanied by severe headache. . . . Four months ago he suddenly lost power in the left arm, and the same evening he became unconscious. Next day he spoke and moved his arm. He noticed the pulsation of his right subclavian artery until four months ago, not since.

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> The right forearm is distinctly colder . . . than the left. . . . The axillary and the third part of the subclavian artery can be plainly felt as a hard pulseless cord. . . .

The sixth reported case of this unusual complication of subclavian arterial thrombosis follows.

Case Report

D. L., a 38-year-old carpenter, was admitted to the Medical Service of the Peter Bent Brigham Hospital on February 3, 1941, with a history of weakness and inability to walk.

Two hours before admission, the patient had noticed sudden weakness of the left hand so that he had dropped his lunchbox twice, felt weak in the knees, and had had to be assisted in walking to his place of employment. Because no improvement occurred after 2 hours, he was sent into the hospital.

Two years previously, he had come to the Out-Door Department complaining of numbness, tingling, pain and weakness in the right hand of 6 weeks' duration. Examination had revealed weak right radial, normal brachial and increased right subclavian arterial pulsations. Weakness and some atrophy of the muscles of the right hand had been noticed, without sensory impairment or decreased skin temperature. X-ray study revealed bilateral cervical ribs. At operation, it was noted that the subclavian artery lay higher than usual, presented a normal caliber and amplitude of pulsation, and did not appear compressed over the rib. Since the brachial plexus also did not appear unduly stretched, it was decided to cut the scalenus anticus muscle and not attempt resection of the rib. Complete symptomatic relief and considerable increase in the amplitude of the right radial pulsation had followed the operation until 6 months before admission, when the patient again complained of mild weakness and occasional paresthesias of the right hand and a decreased right radial pulse. These symptoms had been exacerbated in the 2 weeks prior to admission, and he had in addition noted recurrent pains about the right shoulder and supraclavicular fossa.

The patient had enjoyed excellent health except for a brief attack of neuritis involving both upper arms 16 years before, and an episode of right facial weakness diagnosed as Bell's palsy 12 years prior to this admission.

At the time he was admitted to the ward, neurologic examination revealed a left hemiparesis and weakness of the left lower facial musculature. The deep tendon reflexes were increased on the left. Except for the facial weakness, the cranial nerves were intact. The pupils were equal and reacted well. There was no dysarthria or aphasia. Sensation was nowhere impaired. The Babinski and Kernig signs were absent.

The right radial and brachial pulses could not be detected, and the axillary pulse was greatly diminished. However, pulsation in the right subclavian artery was increased. The blood pressure on the right could not be obtained, but in the left arm it was 120/70, and on this side arterial pulsation was normal. On palpation, no difference in the skin temperature of the two upper extremities could be noted, and there was no evidence of cyanosis or muscular weakness on the right side.

The heart was not enlarged to percussion. The rate was regular, 78 beats per minute, and no murmurs were heard. The remainder of the physical examination was likewise negative.

Laboratory data, which included routine blood, urine and stool examinations, lumbar puncture and skull films, showed no essential deviations from the normal. The Hinton and Wassermann reactions on the blood and spinal fluid were negative.

Following hospitalization, improvement in the neurologic findings was striking. The day following admission, only moderately increased tendon reflexes on the left and a positive Barré sign bore witness to the patient's previous disability.

Five days after entry, however, he began to complain of pain about the right shoulder and numbness of the right hand. Overnight, the right subclavian artery had almost ceased to pulsate, and a hard, tender, fusiform mass replaced the previously dilated and pulsating artery. At the same time, the sedimentation rate, which had been 6 mm. per hour on the day of admission, rose to 34 and 40 mm. on the 5th and 6th hospital days. The oral temperature varied between 98.6 and 99.8°F., falling finally to below 99°F. on the 11th day, when the sedimentation rate reached 19. The numbness and right-shoulder pain gradually improved, the subclavian tenderness disappeared, and a faint pulsation continued to be felt up to the 16th day, when the patient was discharged.

Dr. John Homans, who saw the patient on two occasions, expressed the opinion that sympathetic-nerve irritation from the cervical rib had produced brachial arterial spasm, followed by secondary retrograde thrombosis of the subclavian artery. He advised against surgical intervention at that time, since adequate collateral circulation seemed to have developed. He concurred in the opinion that the cerebral episode was in all likelihood the result of embolism from the thrombus that had formed in the subclavian artery.

Discussion

In this patient, the symptoms of cervical rib had been present for two years and had recurred some months before the cerebral accident. At the time of the embolism, the subclavian arterial pulsation had been present, but an active ascending thromboangiitis may be assumed from the diminished axillary pulsation and the subsequent development of fever, local tenderness and almost complete obliteration of the pulse in the right subclavian artery. The sudden occurrence of left hemiplegia in an otherwise healthy man and its transient character are quite compatible with the diagnosis of a small cerebral embolism. This represents another case in which a thrombus, originating in the right axillary or subclavian artery, propagated itself toward the right common carotid whence a small fragment broke off to lodge in a branch of the right middle cerebral artery and thereby produced a transitory left hemiplegia.

In every reported case of this unusual syndrome, the thrombosis occurred in the *right* subclavian or axillary artery, and the cerebral lesion was also right sided. This is readily explained by the fact that only on the right does the common carotid originate with the subclavian from the innominate artery, so that only on this side of the body would a retrograde thrombus or embolus be caught in the

carotid blood stream and carried to the brain. Should thrombosis occur in the left subclavian artery, emboli would be carried into the aorta and lodge in places less likely to produce distinctive symptoms (Fig. 1). Embolic occlusion of the

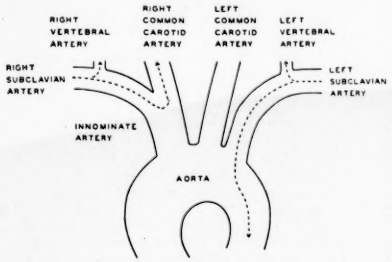

FIGURE 1.

This diagram illustrates the fact that an embolus from a retrograde thrombus in the right subclavian artery is likelier to enter the cerebral circulation than one from a thrombus in the left subclavian artery.

basilar arterial branches, as reported by Yates and Guest,[2] could occur from thrombosis in the right or left subclavian artery, since the vertebral artery on both sides originates from the subclavian. The possibility remains that in the other cases embolism also occurred by way of the vertebral artery, passing by means of the posterior communicating artery into the circle of Willis and, finally, entering the middle cerebral vessels, instead of passing directly through the common carotid artery. This explanation is rendered unlikely by the small caliber of the posterior communicating artery, by the frequency with which the right side is involved, and by the infrequency with which emboli are reported to lodge in other branches of the vertebral artery. Furthermore, in Smith's[3] case, in which autopsy was performed, there was definite proof that the pathway of embolism was the carotid and not the vertebral artery.

The only other findings common to all the cases reported were a history of symptoms of cervical rib or vascular insufficiency over a period of a few months to six years, and an absence of right brachial or radial pulsation at the time of the cerebral accident. In most cases, the subclavian artery had also ceased to pulsate, but in one patient, thrombosis of this vessel did not take place until after the hemiplegia had developed. In no case did definite evidence of ascending thrombosis or thromboangiitis in the right subclavian artery occur immediately prior to the embolism.

It should be emphasized that this complication of subclavian thrombosis is uncommon. In the files of the Peter Bent Brigham Hospital and the Boston City Hospital, no similar cases could be found, and the few isolated reports in the literature bear further witness to its rarity.

Although the conception of embolism originating from retrograde thrombosis of an obstructed artery is somewhat unusual, the occurrence of emboli from mural thrombi located in the wall of large arteries or aneurysms has frequently been reported. Lewis and Pickering[6] suggest that some of the peripheral symptoms of cervical rib may be due to small emboli coming from mural thrombi in the compressed wall of the subclavian artery. In the case reported by Girode et al.,[7] a fatal embolism at the aortic bifurcation was traced at autopsy to a thrombus in an aneurysm of the abdominal aorta. In Marcus's[8] patient, mural thrombosis in the descending aorta gave rise to four distinct embolic episodes. Wood[9] described a young man who complained of pain in the right shoulder, developed suddenly a cold pulseless right extremity, and died fourteen hours later following a left hemiplegia. At post-mortem examination, a walnut-sized aneurysm was found just proximal to the origin of the right common carotid artery, and thrombi were found in the right axillary artery and in the circle of Willis. Unfortunately, the report does not state whether a thrombus was found in the aneurysmal sac, which might have given rise to two separate emboli, whether the axillary thrombus might have been the source of a cerebral embolus, or whether both arteries developed thrombosis in situ.

SUMMARY

A case of cervical rib is described in which retrograde subclavian arterial thrombosis, embolism to the right middle cerebral artery and a resultant left hemiplegia developed. Five cases with a similar syndrome, which have been reported in the literature, are reviewed.

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MEDICAL DISCHARGES FROM MILITARY SERVICE*

A Report of Six Hundred Cases

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CAMP BLANDING, FLORIDA

ARMY requirements provide that, when an enlisted man because of physical disability has become permanently unfitted for military service, he be retained in a hospital until his physical condition has reached such a point that he will not be benefited by further treatment in a military hospital.[1] When he has reached this condition of maximum benefit, a C. D. D. (Certificate of Disability for Discharge[2]) is prepared by his immediate commanding officer and forwarded to the commanding officer of the hospital, station or regiment in which the enlisted man is serving. On receipt of this certificate, the commanding officer convenes a board of medical officers, usually three, to examine the enlisted man critically and to enter on his certificate the required data. These data must include the origin and degree of disability, and must describe particularly the disability, wound or disease, and the extent to which it deprives the enlisted man of the use of any limb or faculty or affects his health, strength, activity, constitution or capacity to labor.

Such discharge is not recommended by this board when in their opinion the enlisted man, although mentally responsible, is inapt or does not possess the required degree of adaptability for the military service, or gives evidence of habits or traits of character that make his retention in the service undesirable, and when the underlying cause for these characteristics is constitutional psychopathic state, moronic state, mental deficiency, chronic alcoholism or drug addiction.§ If, however, it is the opinion of the board of medical officers that the enlisted man is not mentally responsible, he is discharged with a certificate and in accordance to Army Regulation 600-500.

An honorable discharge[3] from the Army of the United States is given if the disability is received in line of duty. If the disability is the result of a man's own misconduct, occurring since or prior to enlistment, he is given a "blue" discharge. This is neither a dishonorable nor an honorable discharge. It is a discharge without honor. If the disability was noted on the enlistment paper, however, or the soldier merits an honorable discharge by reason of honest and faithful service for one or more enlistments since the occurrence of the disease that resulted in the disability, an honorable discharge is given.

It is apparent, therefore, that by the certificates of disability for discharge the Army may dispose of men who have slipped by induction boards and are unfit for military service.

It is also possible to return to civilian life soldiers who, because of mishap, are so injured or afflicted with disease as to be no longer of value in a fighting machine. As a matter of practice, the enlisted man is kept in the hospital wards until a complete and detailed medical workup is obtained. This may, and usually does, include many consultations, laboratory tests, x-ray films and whatever else the staff physicians deem advisable. The physician works with the ease obtainable in pure medical practice; and since no expense is spared, no financial problems are involved. All the medical knowledge, ability and equipment and all the nursing at the command of the hospital are utilized. Every effort is expended to return the soldier to the effective list. However, cases of failure perforce occur. Perhaps the man was admitted to the Army in error. He may not possess the proper qualifications for a soldier. He may have shown, subsequent to induction, epileptic seizures or psychotic or psychoneurotic traits that the inducting physicians were unable to detect. Before induction he may have had flat feet, nonsymptomatic, that subsequently started to cause pain; he may have been a good soldier and been injured with resultant loss of a limb or an eye or he may suddenly be discovered to have diabetes or other serious illness. These are but a few of thousands of possible situations that may necessitate relieving the soldier from military duty.

In Camp Blanding, we have been presented with an opportunity to study personnel from different parts of the country from the time of their induction into the Federal service. We have two divisions: the Thirty-first, or Dixie, Division, comprises troops from Florida, Georgia, Mississippi, Alabama and Louisiana; the Forty-third, or New England, Division, consists of troops from the six New England states. We present herewith the compiled abstracted data on the first 600 men to

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§This group of cases is disposed of according to Army Regulation 615-360, Section VIII.

receive certificates. This survey includes the period from January 15 to July 21, 1941, inclusive.

Rate of Granting Certificates

Figure 1 shows how the rate of certificates has risen since the opening of the camp. The slow on-

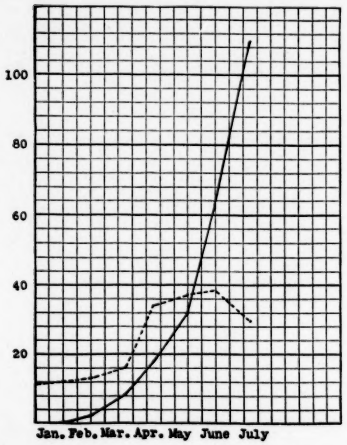

FIGURE 1. *Rate of C.D.D. Discharges.*

The solid line represents the rates of discharge per thousand per annum; the dotted line, the camp strength in thousands.

set, with the sharp rise, must be interpreted as related to the difficulties encountered in getting the hospital routine established and in getting a smooth-

Army regulations for the disposal of casualties. Many certificates were slowed down in the technicalities of regulations. Another factor of considerable weight is that no competent psychiatrist arrived in Camp Blanding until about March 15. This fact also accounts for the high percentage of organic cases in comparison with cases in which the psychic factors were of greatest importance. Of the 600 discharges, 464 disabilities (77.3 per cent) were listed as organic. The diagnoses "flat feet," "sacroiliac disease," "gastritis," and "tachycardia, persistent, etiology unknown," figured largely. Perusal of the charts revealed that many of these cases might more properly have been called neurocirculatory asthenia, gastric neuroses, psychogenic backache and so forth. One hundred and thirty-six men, or 22.7 per cent, were discharged for psychiatric reasons. These included such diagnoses as "mental deficiency" (42), "psychoneurosis" (63), "psychosis" (23) and others (8). Subsequent figures have shown that the discharges from psychiatric causes in a peacetime army may even exceed those from organic causes.[4]

Age

Despite popular conception to the contrary, the number of discharges did not increase with age, and the ratio of certificates to the number of men in each age group was quite constant. Figure 2 shows the actual number of men discharged in each age group. Figure 3 shows the percentage of discharges of each age group. This is compared with the percentage of men of each age in a random sample of 3000 soldiers.

Duration of Service

How long is it before a soldier manifests signs indicating his inability to fit into the military or-

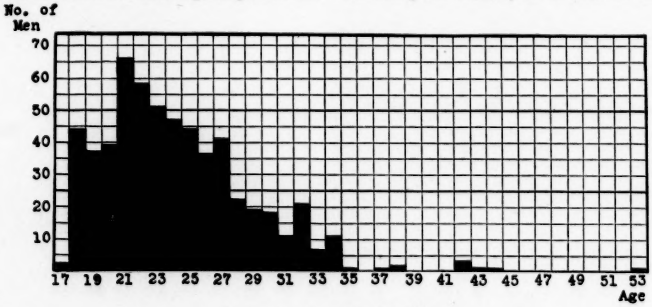

FIGURE 2. *Age When Discharged.*

ly functioning organization for the disposal of patients entitled to certificates. It must be remembered that the divisions involved were National Guard divisions that had insufficient knowledge of

ganization? How long does it take the Army to determine that a man is unfit to be a soldier? Figure 4 shows our findings. The significant fact is that the peak is at three months. Since in most cases

about six weeks elapse in the machinery of obtaining a certificate (complete hospital workup, holding of a board and obtaining of necessary endorsements), it can readily be seen that most men who are going to break down do so rapidly and are rather promptly recognized. Furthermore, in the reported cases, we believe that the lag after three

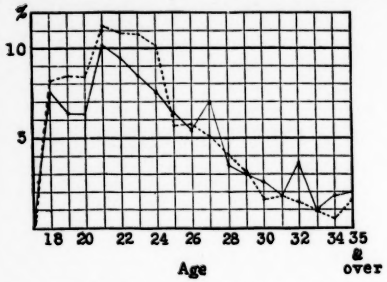

FIGURE 3. *Percentages of Discharge by Age.*

The solid line represents the percentages of discharge by age; the dotted line, the percentages of camp population by age.

months is attributable to the newness of the divisions in Federal service and the slowness in the establishment of the routine for obtaining certificates. It must be borne in mind that these divisions until quite recently were in the service of the various states and that the men trained one or two nights per week and, possibly, three weeks during the summer each year. They had, therefore, a limited experience in the methods of the Army.

Previous Occupation

It was rather difficult to arrive at any definite conclusions regarding previous occupations. In the first place, each grouping had to be quite arbitrary. Whether a farmer is to be considered an unskilled, semiskilled or skilled laborer is a moot point, and whether a truck driver who handles a trailer truck is unskilled or skilled is equally debatable. However, these questions were solved as seemed justifiable in each particular case. College students, lawyers, veterinarians, a physician (who was discharged for epilepsy and later committed suicide), a doctor of philosophy in chemistry and a chemist were in the professional group; merchants, store clerks and salesmen were placed in the business group. Table 1 shows the number of men in each occupational group who were discharged, the percentage each group contributed to the 600 discharges, and the percentage of men in each occupational group as taken from the previously mentioned sample of 3000 soldiers. It is apparent that the more educated and highly trained the person, the better his adaptability and the greater the likelihood of his being a successful soldier. It reminds one of a statement attributed to General Pershing, "The best citizens make the best soldiers." Furthermore, there was a considerable difference in the reasons for the failures of the educated and uneducated.[4]

TABLE 1. *Occupations of Men Receiving Certificates of Disability for Discharge.*

OCCUPATION	No. DISCHARGED	PERCENTAGE OF TOTAL DISCHARGES	PERCENTAGE OF 3000 SOLDIERS
Unskilled labor	234	37.0	28.7
Semiskilled labor	103	17.1	23.1
Skilled labor	131	21.8	20.2
Business	90	15.0	18.4
Professional	42	7.0	9.6

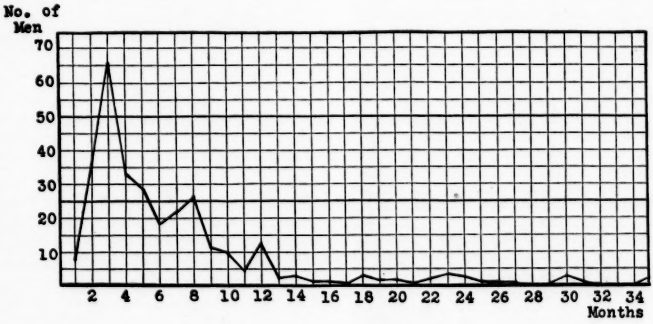

FIGURE 4. *Months of Service before C. D. D.*

Rank

Among the noncommissioned officers, selection, both psychologic and physical, had already taken place to a great extent. Those who were dis-

charged usually had quite a duration of service and received certificates for physical disabilities that were frequently engendered in line of service. Very few of these noncommissioned officers were discharged with such diagnoses as "psychoneurosis." They were less frequently discharged, from a percentage basis in relation to their numbers, than the privates, and the ratio decreased with the increase in rank. This is quite obvious in Table 2, which shows the number of men in each grade discharged, the percentage of the discharges in each rank and the average percentage of men in each rank in the Army as a whole.

TABLE 2. *Ranks of Men Discharged.*

RANK	NO. DISCHARGED	PERCENTAGE OF TOTAL DISCHARGES	AVERAGE PERCENTAGE IN ARMY
Private	479	78.5	52.0
Private, first class	57	9.5	30.0
Corporal	38	6.3	10.0
Sergeant	26	4.3	8.0

Type of Service

This camp is essentially an infantry camp, possessing only one field-artillery brigade and no cavalry units. Other troops include military police, a hospital detachment, a quartermaster corps and similar corps-area-service command troops. No significant relation could be found between the number of discharges and the type of military service performed, these figures standing approximately in direct ratio to the number of troops engaged. Thus, 400 certificates were contributed by the infantry, 88 by the artillery, and 112 by other groups. The type of military service could not be regarded as a contributory factor to medical discharge.

Urban versus Rural Population

Arbitrarily selecting all municipalities listed in the *1941 World Almanac* that contain 2500 inhabitants or over as representing urban life, we divided the men in accordance with rural or urban origin (Table 3). The percentage of discharges among men from rural sections was all out of proportion to the percentage of such troops in the Army, and leads one to the assumption that the "stalwart farmer boy" may not be so good a soldier as his city brother.

TABLE 3. *Discharges among Men from Urban and Rural Sections.*

SECTION	NO. DISCHARGED	PERCENTAGE OF TOTAL DISCHARGES	PERCENTAGE OF 3000 SOLDIERS
Urban	108	18.0	32.4
Rural	492	82.0	67.6

Incidence by States

On July 25, the comparative strength of the two divisions was: the Thirty-first, 17,942 men, and the Forty-third, 16,483 men. By that date, there had been 149 certificates from the Forty-third and 410 from the Thirty-first Division, 41 being listed as from "other troops." Reduced to percentages, it had become necessary for the Thirty-first Division to discharge 2.29 per cent of its personnel whereas the Forty-third Division had released only 0.84 per cent. The number of cases by states is shown in Table 4. These are the states in which the men were inducted into military service.

TABLE 4. *Discharges by Sections and States.*

SECTION	TOTAL NO. OF MEN	NO. OF DISCHARGES
New England (43rd Division)	16,483	149 (0.84%)
Maine		20
New Hampshire		4
Vermont		31
Massachusetts		1
Rhode Island		11
Connecticut		82
South (31st Division)	17,942	410 (2.29%)
Georgia		17
Florida		130
Alabama		81
Mississippi		90
Louisiana		92

Line of Duty

Of the 600 cases reported, only 8 were marked as "line-of-duty" discharges. In all the others, the notation was made that the disease or disability "existed prior to enlistment."

SUMMARY

An analysis of the first 600 cases relieved from duty at Camp Blanding under the Army regulation providing for certificates of disability for discharge is presented. The rate of discharges, the relation of organic to psychic factors as causes of disability, the age of enlisted men at the time, the duration of service prior to medical discharge, the relation of previous earned rank and type of service, whether the cases were urban or rural, and, finally, the geographical distribution by sections and states are given.

These statistics were collected during the formative period of the camp, and further studies may show substantial changes in some of these factors.

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4. Unpublished data.

ANTHRAX: REPORT OF A FATAL CASE INVOLVING THE CUTANEOUS AND GASTROINTESTINAL SYSTEMS*

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HUMAN anthrax, usually involving the skin, is still an industrial hazard confronting workers in tanneries and wool industries and, to a certain extent, agricultural laborers. Since most industrial anthrax is of the cutaneous type and the reports of internal anthrax (pulmonary and gastrointestinal) in the literature are so few, the following case is presented.

CASE REPORT

H. S., a 38-year-old man, was admitted to the hospital on June 17, 1941, obviously dangerously ill. He had been employed for years in a Worcester brush factory, where he handled bristles imported from China, India and Manchukuo.

Four days before admission, the patient was seen at home by his physician, who treated him for sore throat and swelling of the lymph nodes on the right side of the neck. He did not seem acutely ill at this time. However, he appeared much worse each day of his illness, and a day prior to admission, his face was markedly swollen, the pulse was of poor quality, and the temperature was subnormal. Moreover, the abdomen was tense and distended and very tender to pressure.

On the morning of admission, the patient's condition grew critical at home, and he was referred to the Belmont Hospital with a tentative diagnosis of mumps. Meanwhile, a pustule on his forehead aroused the suspicion of anthrax.

On admission, the patient's temperature was 97.0°F., and no pulse could be detected at the wrist. The respirations were 40 per minute and very labored. The patient was drenched in perspiration, the lips were cyanotic, and there was a generalized, mottled cyanosis of the skin. He was rational and co-operative, and complained of abdominal pain, inability to catch his breath and weakness. On the forehead, just above the right eyebrow, was an area of redness about the size of a five-cent piece. This lesion was raised and indurated. Topping the lesion was a yellow pustule with a central umbilicated hemorrhagic and necrotic area. The tongue was dry and heavily coated. The roof of the mouth was studded with eight to ten tiny yellow pustules. The pharynx was red and injected, and a heavy, greenish-yellow exudate was noted in the postnasal space. There was a large brawny induration of the right side of the face and neck. This mass was hard, and no pitting edema or fluctuation could be felt. The heart sounds were weak, — barely audible, — and no murmurs were heard. The blood pressure could not be recorded. Nothing abnormal was found in the lungs. The abdomen was tense, tympanitic and markedly distended. There was tenderness, with spasm, over the entire upper abdomen. No palpable masses or viscera were felt. The extremities were mottled in appearance and very cold to palpation. There were no neurologic changes.

The diagnosis on admission was: anthrax, cutaneous type; ? gastrointestinal anthrax; and ? anthrax bacteremia.

The diagnosis was tentatively confirmed by direct smear of the purulent material from the lesion on the forehead. The blood culture taken on admission showed a heavy growth of *Bacillus anthracis* in 36 hours.

The patient was immediately treated for shock. He was given 1000 cc. of 5 per cent glucose by vein and shortly thereafter 2 gm. of sodium sulfapyridine, also by vein. As soon as a suitable donor could be obtained, the patient received 500 cc. of citrated blood. Meanwhile, a request was sent to Boston for 300 cc. of specific antiserum. After the transfusion, the patient improved for a while, but the pulse did not become perceptible. He was catheterized because of inability to void for about a day at home, but only a small amount (20 to 25 cc.) of very concentrated urine was obtained.

Examination of the blood showed a red-cell count of 5,500,000 with a hemoglobin of 90 per cent (Sahli), and a white-cell count of 10,600. The urine showed a specific gravity of 1.023, acid reaction, the slightest possible trace of albumin and no sugar. The sediment showed a few hyaline casts and a few red and white blood cells per high-power field. Smears and cultures of the lesion on the forehead were positive for *B. anthracis*. The nonprotein nitrogen was 69 mg. and the blood sugar 143 mg. per 100 cc.

The patient became progressively worse; he developed delirium about 5 hours after admission, and died 1 hour later, before serum could be obtained. Prior to expiration, the patient vomited a large amount of coffee-grounds material mixed with bright-red blood, and he had a terminal rise in temperature to 103.0°F.

Autopsy (performed by Dr. Raymond Goodale of the Worcester City Hospital). There was an elevated skin lesion just above the right eyebrow. This lesion measured 1.2 cm. in diameter, with a 3.0-mm. central depression. In addition, a few golden-yellow lesions were noted on the hard palate.

The lymph nodes on the right side of the neck were enlarged, and the surrounding edema extended down to the clavicle on that side. The organs of the chest were grossly normal.

The abdominal cavity contained about 500 cc. of cloudy, amber-colored fluid. The mesentery contained a few hemorrhagic areas in which the fat was swollen so that it measured 5 cm. in thickness.

The thoracic esophagus was normal. The stomach appeared grossly normal but contained 50 cc. of coffee-grounds material. Just below the ampulla of Vater was an edematous area in the mucosa measuring 6.0 cm. in diameter and giving the intestinal wall a total thickness of 1.5 cm. The lesion was red, and there was a glistening mucinous appearance on section. In the lower jejunum, 170 cm. from the duodenum, was the second lesion, which measured 1 cm. in diameter. The third lesion, located 200 cm. above the ileocecal valve, measured 1.5 cm. in diameter. The fourth lesion was 190 cm. above the ileocecal valve and also measured 1.5 cm. in diameter. The fifth and largest lesion, which was located 170 cm. above

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the ileocecal valve, measured 18 cm. in length; it was intensely red and swollen, and the mucosa showed signs of necrosis (Fig. 1). The final lesion was located 120 cm. from the ileocecal valve, and was 15 cm. long; the mucosa was pink and swollen but no hemorrhage or necrosis was present. All lesions presented a mucinous appearance on section.

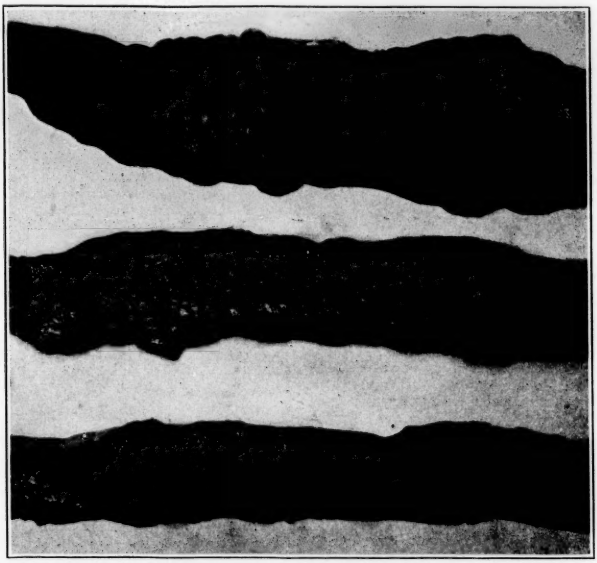

FIGURE 1. *Lesions of the Small Bowel.*

The upper specimen is a section of small bowel showing maximum involvement, with edema, hemorrhagic changes and early necrosis; the middle one shows the earlier changes, which consist of edema, a gelatinous exudate and some hemorrhagic changes, with no necrosis. The lower one is a section of normal small bowel.

Anthrax bacilli were recovered from the cultures of the pus from the lesion on the forehead, of the free abdominal fluid and of the blood removed from the right auricle.

Microscopic examination of the mesenteric lymph nodes showed an acute, hemorrhagic adenitis; there were acute inflammation and edema of the ileum, a few anthrax bacilli being present.

DISCUSSION

This case represents a massive inoculation with *B. anthracis*. The cutaneous lesion occurs usually after contamination of an apparently healthy skin with spores of the anthrax bacillus. In the case presented, there was no history of previous trauma to the skin. Most writers, however, postulate a break in the skin before infection can take place.

The mode of invasion of the gastrointestinal tract was undoubtedly the swallowing of large numbers of the spores. Lucchesi[1] points out that the mouth may be infected by contaminated hands.

The fatality rate from cutaneous anthrax is still high, 16 per cent being average mortality for the cases reported during the five-year period 1934–1938. However, this represents a 6 per cent reduction from the previous five-year period. Internal anthrax, on the other hand, is almost always fatal. Eurich[2] reports 23 cases of pulmonary anthrax that ended fatally. He had seen but one case of gastrointestinal anthrax. Moreover, the recent literature does not report a single case of recovery from gastrointestinal anthrax. There is a possibility, however, that the outlook for internal infection will improve with newer methods of treatment, as evidenced by the recent report of Lucchesi and Gildersleeve,[3] who report 2 cases of recovery following anthrax bacteremia. Moreover, one of the cases was of the pulmonary type, which is usually rapidly fatal.

In the treatment of anthrax, several features stand out clearly. The recent investigators are apparently in agreement that local incision and excision of the anthrax pustule is contraindicated unless the lesion is very small and easily accessible for surgery. Moreover, the practice of local injection of serums around the lesion has been discontinued in most clinics.

Serotherapy, given intravenously and occasionally intramuscularly, seems to be indicated in all cases of anthrax. The method used is fairly large doses (100 to 300 cc.) given at twenty-four-hour intervals over a three-day period.[3] Neoarsphenamine given in 0.6-gm. to 0.9-gm. doses over this same three-day period has proved to be a valuable adjunct in treatment[3]; this may permit reduction in the dose of serum.

Recently, sulfanilamide and sulfapyridine have been used in the therapy of anthrax. In guinea pigs, according to Mitchell, Walker and McKercher,[4] sulfanilamide seems to control the spread of the anthrax infection; however, this drug has not been used in a sufficient number of human cases to provide definite conclusions of its value. Bonnar[5] reports recoveries in 2 critical cases in which sulfapyridine in conjunction with serum and neoarsphenamine was used.

Summary and Conclusions

A fatal case of internal anthrax with bacteremia is reported, with autopsy findings.

Anthrax still remains an occupational hazard to workers in wool, hair and hides, in spite of rigid control of imported materials.

The prognosis of internal anthrax is grave in spite of the excellent results obtained in the therapy of cutaneous anthrax with specific serum and neoarsphenamine.

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MEDICAL PROGRESS

VASCULAR DISORDERS OF THE EXTREMITIES (Concluded)

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BOSTON

Post-Traumatic Pain Complexes

UNDER the titles "post-traumatic pain syndromes" (Livingston[37]), "reflex dystrophy of the extremities" (de Takats[38]), "trophoneuroses" and many others, these peculiar painful states have long been looked at askance by the profession in general as functional, perhaps hysterical, disorders. Recently, Homans[39] has discussed them under the title "minor causalgia" and has called attention to the fact that similar disorders may follow inflammatory thrombophlebitis of the legs. That painful, edematous states associated with paresthesia of the skin may arise from thrombophlebitis tends to confirm earlier impressions that such syndromes represent a vicious reflex arising from irritated perivascular sensory-nerve fibers.

The minor causalgias are usually insurance problems and as such have considerable economic importance. The initial injury is usually trifling. A blow on the hand, forearm or foot, the wound of a thorn, a burn, a frostbite, a minor fracture or sprain, a felon, a "milk leg," any of these may establish the reflex disorder. The hand is oftenest affected. Some part of it, usually a roughly anatomic field of one or more great nerves, is "dull" to pinprick, cool to the touch, slightly edematous (knuckle markings lost) and faintly bluish. The part feels "stiff," the grip is lost, and there may be some sensitive area — trigger point — that causes the patient to protect the part from all contacts. The severity and persistence of the causalgia vary greatly, but a hysterical element is rarely present and most of the victims desire to work.

The causalgia can usually be cured by the breaking up of the reflex, even temporarily, by the interruption of sensory impulses at the trigger point (Livingston), on the principal artery leading from the part, in the somatic nerve serving the part (Livingston) or in the sympathetic chain at the root of the affected limb. A single injection of procaine may cure. Repeated injections have a progressively good effect. Gentle measures and persistence are usually rewarded. Probably, surgery should not go beyond periarterial sympathectomy or paravertebral sympathectomy, al-

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though excision of a sensitive scar or sinus is sometimes successful.

Thrombosis in Deep Veins of Extremities

Within the last few years, the venous thromboses in the lower limbs have been separated into two classes: the quiet, nonobstructive, embolus-threatening sort, or *phlebothrombosis* (Ochsner and DeBakey[40]), and the outspoken, inflammatory, obstructive sort, or *thrombophlebitis*. The pathological studies of Neumann,[41] Frykholm[42] and Hunter et al.[43] have proved that in fatal cases (pulmonary embolism) the thrombosis, almost necessarily the quiet phlebothrombosis, originated in the venous plexuses among the muscles, particularly in those of the calf but sometimes in those of the foot or thigh. Venographic examinations, instigated by dos Santos[44] and greatly developed by Bauer,[45] have shown how the common, quiet process, complicating injury, operation or disease, or even appearing during active life (Homans[46]), may remain local or progress into the femoral vein. Here it may form a long, loose, fatal embolus or work its way centrally in a lightly adherent, nonobstructing form into the upper femoral or external iliac vein, or actually fill and block the latter vein, causing the familiar phlegmasia alba dolens of thrombophlebitis.

Phlebothrombosis

This relatively quiet process may be utterly silent until a serious embolism occurs. Or it may cause lameness (on walking), pain in the calf, full, slightly tense, tender muscles, discomfort on forced dorsiflexion — the dorsiflexion sign — and a varying degree of edema and cyanosis, which may be absent on elevation and only evident on dependency.

There is some difference of opinion concerning treatment. The statistical figures of Barker, Nygaard, Walters and Priestley[47] indicate that about one person in twenty in whom thrombosis is *recognized* will suffer an embolism whether or not fatal; and that one person in five, having experienced a pulmonary infarction, will die of a later embolism. As a result of their clinical experiences, Fine and Sears[48] recommend, as a routine procedure, exploring the femoral vein, to head off the embolism, remove the clot and so forth, whenever a diagnosis is made. Welch and Faxon[49] favor it. Welch, Faxon and McGahey,[50] by combining the routine use of venography with special clinical observations, have determined to their satisfaction the indications for femoral exploration and the management of thrombosis at various levels and stages. Homans[51] prefers conservative treatment in early cases, but opens and divides the femoral or one of the iliac veins when embolism or recurrence of symptoms (after nonoperative treatment) indicates that healing without further accident is unlikely. For conservative treatment, he recommends 6-inch elevation of the foot of the bed, freedom of movement, no ice bag, no bending at the hip and, after ten to fourteen days of such elevation, gradual resumption of activity in a semielastic bandage.

The value of heparin is difficult to assess. Murray and Best[52] have shown that routine postoperative heparinization heads off thrombosis. Murray[53] points out that in a considerable series of cases, heparinization, instituted only after pulmonary infarction had already occurred, prevented further embolism* (an important observation in view of the percentage of deaths following a preliminary infarction recorded by Barker et al.[47]). Heparin may be given as a continuous intravenous drip (Murray and Best) or every four hours, in a dosage of about 60 mg. of purified heparin, constantly checked by the clotting time (capillary-tube method). The expense is at least seven or eight dollars a day even without special nursing. When surgery is not available and especially when expense is not a problem, heparinization seems indicated. A clotting time of ten to twenty-five minutes should be maintained for three to five days and then tapered off, to avoid the danger of recurrent thrombosis.

Dicoumarin, whose use is in the experimental stage, has recently been put forward as an anticoagulant. Provided it is efficient and not harmful, the fact that it can be given by mouth gives it an advantage over heparin. Unlike heparin, whose action can be stopped immediately on neutralization by protamine, dicoumarin, once in the circulation, cannot be neutralized, and transfusion of blood is not always a satisfactory means of control of the dicoumarin effect. Dicoumarin lengthens the clotting time materially only after being administered for several days, and its action continues for several days after it has ceased to be given (Davidson[54]). Thus, present information indicates that caution should be used in the acceptance of dicoumarin as a satisfactory substitute for heparin in such situations as those for which heparin is now known to be valuable.

Thrombophlebitis

Irrespective of its origin, that is, whether it begins in a thrombosis low in the leg, in the uterine veins or locally in the femoroiliac region, thrombophlebitis, phlegmasia alba dolens or "milk leg" is an obstructive process in which an inflammatory exudate often surrounds the femoral and iliac veins

*Although no figures comparable with Murray's are available, deaths from embolism under similar circumstances have occurred in very much smaller series in the experience of other surgeons.

and arteries. Some degree of local arterial spasm, as already noted, results. (Experimentally, the effect on arterial blood flow of a local irritation within and about a large vein is described by DeBakey, Burch and Ochsner.[55]) Also, as a reflex, a diffuse peripheral vasospasm in the arterioles or venules, or both, perhaps, occurs. The resulting edema is due partly to venous obstruction, partly to lymphatic obstruction and partly (in some cases almost exclusively) to the peripheral vasospasm. This last has been Leriche's[56] contribution, and following his observation that lumbar sympathetic procaine block relieves the edema in a remarkable way, Oschsner and DeBakey[57] have fully discussed the pathologic physiology and have popularized this treatment.

The special indications for making use of a sympathetic block are edema, fever and discomfort (as signs of a perivascular irritation). But routine conservative treatment should still be carried on. This consists in elevation of the foot of the bed, without immobilization of the leg, and the use of a heated cradle, to promote vasorelaxation. Ice bags should not be applied. Sympathetic block is most effective if used early. The technic is based on one or another of Labat's[58] methods of securing a paravertebral anesthesia, but adapted by White[59] to reach the sympathetic chain just ventral to the lateral curve of the vertebral body.

Ochsner and DeBakey[57] introduce three needles (4-inch lumbar-puncture needles, with stylets) through wheals placed respectively about two fingerbreadths (roughly, 4 to 5 cm.) lateral to the midline of the back and opposite the first, second and third lumbar interspaces. Each needle, entering perpendicularly to the plane of the back, should strike a transverse process at a depth of about 4 or 5 cm. The needle is then redirected above or below the process and somewhat medially, meeting next the lateral border of the vertebra. Another readjustment in a more lateral direction causes its point to glide past the vertebral belly for perhaps 1 cm. into the region of the gangliated chain. Five to ten cubic centimeters of a 1 per cent procaine solution is injected through each needle.

On the basis of Flothow's[60] recommendation of Lundy's[61] method, others (Reichert,[62] de Takats[63] and Homans[64]) prefer to introduce a 4-inch (for women) or 5-inch (for men) needle, with stylet, 7 cm. lateral to the midline opposite the first lumbar interspace. This spot is near the twelfth rib and the outer margin of the erector spinae muscles. The needle is directed toward the midline at an angle of about 35° from the perpendicular. A transverse process may or may not be met at a depth of 4 or 5 cm., the plan being to strike the broad lateral curve of the vertebra at a deeper level. The needle is then partly withdrawn and redirected more laterally so that it glides just past the vertebral belly. This method increases not only the chances of placing the needle point close to the sympathetic chain, which lies toward the front of the vertebra, but also the risk of injuring the aorta (on the left) if the needle is thrust too deeply. However, if it is passed no more than 1 cm. beyond its last bony contact and meets no resistance in that last step, an arterial injury is extremely unlikely. Suction should always be made, however, before injection, to make sure no vessel has been entered. If any bleeding occurs, the needle should be withdrawn and reinserted at a higher or lower level.

Postphlebitic pain complex. Postphlebitic pain, associated with paresthesia below the knee and some degree of edema and cyanosis, may remain after a femoroiliac thrombophlebitis, a causalgia-like state (Homans[39]). Lumbar sympathetic block, performed during the acute stage of thrombophlebitis, tends to ward off this sequel. In many cases, one or more sympathetic blocks often cure the pain complex itself. The favorable effect of one block should not be allowed to lapse before the next block is given. Thus, in time, even an obstinate process may often be relieved. If not, sympathectomy combined with lysis of adhesions between the iliac vessels on the affected side — or even resection of the diseased vein — is likely to succeed (Leriche and Geisendorf[65]).

Varicose Veins

Etiologic Factors

The causative factors of varicose veins have been studied by Adams[66] from the point of view of venous pressures and the effect of valvular incompetence. By means of direct-pressure determinations in the normal great saphenous vein, Adams found that the presence of normal valves did not alter the transmission of pressures in the fluid system and that such pressures followed Pascal's law: namely, that "pressure applied to an enclosed fluid is transmitted equally in all directions and acts with equal force on all surfaces." Thus, pressures at any one level represent the weight of the column of blood between that level and the heart. Adams points out that when the patient is upright the resulting high pressures in the leg tend to overstretch the superficial veins unprotected by surrounding muscle. So long as valves remain competent, however, additional pressures from above, caused by straining and lifting, can raise pressure within the saphenous system little if any higher than gravity itself can do. But once the valves become incompetent, the effect of straining and lifting is to raise superficial venous

pressure in varicose veins to pathologic levels. In such veins, as is well known, pressure may rise to high arterial levels. Adams then makes some interesting inferences:

> "Recannulization" (Adams's word) will occur after treatment by injection unless a high preliminary division is carried out. High division should not be condemned because pressures in the remaining varices below are not lowered, for all that high division can do is to prevent the additional elevation of pressure due to straining and lifting.

Adams advises the inclusion, in the Trendelenburg test for varicosity, of a consideration of the additional effect, beyond gravity alone, of straining.

Veal and Hussey[67] have made direct examination of pressures in the popliteal veins of pregnant women. They studied especially the effect of exercise to be sure that the engorgement of exercise should be given consideration. As between subjects having respectively normal and varicose (or edematous) legs and between the normal and varicose leg of one pregnant woman, popliteal pressures remained stationary on exercise in the normal leg and rose decidedly in the varicose. They conclude that local venous obstructions within the pelves of some pregnant women have led to varicosities by causing pressures higher than normal in the veins of the legs and that, in selected cases, support of the superficial veins during pregnancy by an elastic stocking or bandage should be advantageous.

Edwards and Edwards[68] have continued their interesting observations on the valves of the veins in the legs. In earlier communications, they[69] discussed the orientation of valves in relation to the body's surface, and the destruction of valves by thrombosis. Now they show how overstretching of the vein's wall causes valves to become useless. They present models of cross sections.

Tests for Varicosity

There is an evident tendency in some clinics to make rather elaborate tests of varicosity. Mahorner and Ochsner[70] have developed elaborations of the Perthes test (observation of the state of filling of superficial veins below an elastic constriction during exercise) to demonstrate obstruction or normalcy of the deep veins and also the presence of incompetent perforating veins at some particular level. So far as the situation of incompetent communicating veins is concerned, the tests of these authors, if intelligently and skillfully performed, show the need for accurate dissection and division of perforating veins in particular patients and particular localities. Experts can well use them for this purpose. (It should be noted that elaborate tests of deep venous obstruction in the presence of varicose veins are unnecessary. For since varicose veins will not transmit blood upward in the standing position, a normally colored foot in the dependent varicose leg on standing or exercise guarantees a normal deep venous return. Moreover, blocking or removing varicose veins should *always* lighten the load of the deeper system irrespective of the state of that system.)

Pratt[71] regards undiscovered and undivided incompetent communicating veins as a common cause of recurrence after high division and retrograde injection of varicose veins. He considers the Mahorner–Ochsner tests to be "difficult to perform and to interpret and . . . so time consuming that they were not useful in the clinic." He proposes a test that uses the constricting rubber tube about the upper thigh to prevent down-flow through the saphenous vein and applies a bandage from the toes up to the level of this constriction. As the leg is lowered and this bandage is removed by degrees from above, the location of incompetent communicating veins or "blowouts" is revealed. He recommends that such blowouts be surgically corrected. (It is questionable whether the incompetent communicating vein, which Pratt and others describe as a "blowout," affords so direct and open a leak from the deep veins to the varicose superficial ones as is pictured. Anatomic studies made many years ago [Jarjavay[72]] show that alternative routes between the deep and superficial systems are available and are usually able to conduct blood in or out according to local pressures in one system or the other. Thus, although the communicating veins normally conduct blood from surface to depth, some, or all, of them are subject to local pressure influences, through the action of by-passes. Undoubtedly, attention *should* be given to gross leaks outward into varicose veins, and these ought to be considered in planning surgical treatment. But normally high pressures in the postoperative varicose system are consistent with competent as well as incompetent communicating veins. In this connection, Adams's[66] communication is significant, and pathologically high pressures, that is, on straining and coughing, are far likelier to be re-established, in that part of a varicose system left below a high division, by a reconnection of varicose vessels at the groin than by the communicating veins of the lower leg.)

Elephantiasis

A very ingenious and useful contribution to the surgery of elephantiasis has been made by Macey.[73] In a patient with a great congenital hemangiolymphangioma of the arm, a procedure involving the burying of a split graft in the muscle of the fore-

arm and beneath the greatly deformed lymphangiomatous, superficial tissues was attempted. At the end of two weeks, the superficial parts were dissected from the surface of the graft, which had healed perfectly. Only a little loose connective tissue was left between the skin graft and the muscle. Evidently, the forearm and the back of the hand were treated in two stages, although the operator, in describing the first operation, makes the almost incredible statement that the "lymph-edematous, subcutaneous tissue was dissected from the musculature of the forearm in a completely encircling manner."

Undoubtedly, the principle can be applied to the not very uncommon, disabling elephantiasis nostra, or praecox, of the leg, particularly by the aid of the huge and uniform "half" and "three-quarter" thickness grafts that Padgett's[74] machine is capable of cutting. If such means prove suitable for covering the deep parts of the whole lower leg, the ankle and the front of the foot, they should prove superior to the procedure of replanting great attached flaps of pure skin on the subaponeurotic structures (Homans[75]), by which half the lower leg and foot can be treated at a sitting. Similar plastic flap operations have been used by Knott,[76] who has had a wide experience with the tropical form of the disease. Knott has also devised most ingenious ways of compressing, by various bandaging devices, the elephantiac leg and finds that, after the leg has been reduced to an approximately normal size, not only are the inflammatory attacks eliminated but a plastic operation is made relatively easy.

Macey's communication, valuable as it is, contains one or two statements that can hardly be allowed to pass unchallenged. He states:

> The original conception of the Kondoleon operation, that of establishing a communication between the superficial and deep lymphatics, has been discarded and it is now felt that the superficial lymphatics drain directly into the vascular network of the muscles after removal of the deep fascia. . . . On microscopic examination sections of normal muscle have been demonstrated to have abundant lymphatics and from the clinical observation, the lymphatics in the muscles seem uninvolved by the lymphedema.

As to the first of these statements, there is no evidence of any sort that, as a result of a plastic operation, fluids collected between the skin and the deeper structures are carried into spaces between the muscles or that such vascular network as may be present can absorb the highly proteinized tissue fluids of elephantiasis. And as to the second, namely, that abundant lymphatics exist in muscle, there are, it is true, some lymphatics in the intermuscular fibrous tissue of normal persons, but that lymphatics exist in the muscle proper, there is no convincing proof (Drinker and Yoffey[77]). Apparently, in fully developed elephantiasis, no valved lymphatics are left in the limb. If any tissue fluid passes centrally between the muscles, it must be forwarded by muscular squeezing and pumping. It is not necessary that subaponeurotic lymphatics be present to make a radical plastic operation safe in elephantiasis. In the elephantiac leg, fluid passes through wide spaces in the fibrosed superficial tissues by gravity alone. Such tissue fluids as remain after most of the subcutaneous tissues have been excised probably drift between skin and the underlying muscle, or even in the skin itself, as elastic pressure and gravity direct. Some years ago, Gillies and Fraser[78] implanted pedicled flaps containing lymphatics (from the arm) on the elephantiac leg, with the idea of draining its fluids into a part of the body where absorption was normal. Recently Pratt and Wright[79] have proposed a similar procedure. Even aside from the technical difficulty and dubious success of such elaborate plastic operations, the plan of excising the fluid-bearing, subcutaneous tissues seems decidedly more practical.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28241

PRESENTATION OF CASE

A fifty-three-year-old, single female office worker was admitted to the hospital because of jaundice of a month's duration.

About ten years before entry, the patient began to suffer from recurrent attacks of "indigestion," characterized by belching, epigastric distention, nausea and vomiting. These spells, which tended to occur after meals of fatty foods, continued sporadically for about six years, although the patient went on a low-fat diet, on the advice of her physician. In the three years preceding entry, she remained quite well, and gradually returned to a full diet. Two months before entry, she experienced a constant sense of pressure in the epigastrium, lasting for about two weeks. This feeling was aggravated by deep respiration and was not accompanied by any other symptom. A month before entry, the patient noted that her stools were clay colored. In the next week, generalized pruritus appeared, and the urine became deep orange yellow. She was given "vitamin K and bile salts" and was put back on a low-fat diet. The symptoms continued to progress, with the addition of weakness and lassitude. The appetite was only fair in the month preceding entry, and in this time she lost 14 pounds in weight. At no time was there nausea, vomiting, pain, chills or fever.

A younger sister of the patient had had an operation for "gall-bladder disease," and a brother had died in childhood of typhoid fever. The patient thought that she herself had had a transient attack of jaundice as a child. She denied the use of alcohol and of drugs other than those specified. Six months before entry, she had a normal menopause.

On admission, the patient appeared well developed and nourished, and in some distress from diffuse pruritus. There was evidence of recent weight loss. The skin and scleras were icteric, and all over the body there were multiple excoriations from scratching. The heart and lungs seemed normal. A smoothly rounded, slightly tender mass, approximately 5 cm. in diameter and fixed on respiration, lay in the right upper quadrant of the abdomen. The liver edge was not felt.

The blood pressure was 148 systolic, 105 diastolic. The temperature, pulse and respirations were normal.

Examination of the blood showed a red-cell count of 3,960,000 with 11.3 gm. hemoglobin, and a white-cell count of 4600 with 72 per cent polymorphonuclears, 12 per cent small lymphocytes, 12 per cent monocytes and 4 per cent eosinophils. The blood Hinton reaction was negative. The van den Bergh reaction was biphasic, with 24 mg. bilirubin per 100 cc. of serum. The hematocrit was 40 per cent. The prothrombin time was elevated to about four times normal. The serum protein was 6.2 gm., and the nonprotein nitrogen 28 mg. per 100 cc. The blood sugar was 70 mg. per 100 cc. The urine was dark amber, with a ++ test for bile pigments. The stools were clay colored, with negative reactions for bile pigment and for occult blood.

The patient was given Klotogen, Bilein and Hykinone, with a fall in the prothrombin time to normal. The temperature remained normal. On the seventh hospital day, an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. WYMAN RICHARDSON: To summarize this case very briefly, the patient for ten years had symptoms consistent with gall-bladder disease, and then developed an obstructive jaundice and right-upper-quadrant mass. One is forced to make a diagnosis of cancer involving probably the gall bladder on the basis of pre-existing cholelithiasis. I should stop there. I shall go on, however, and add one or two things. In the first place, I had a case like this a year ago — in a fifty-three-year-old woman with a similar history — in which an indefinite right-upper-quadrant mass was disregarded because the patient was obese. I should have paid attention to it because she had a tumor of the biliary tract. In the case under discussion, the mass was definite, and I wonder if I am going to be misled about it again.

One can assume this mass to be gall bladder. One can also assume that the patient had gallstones and that she had a gallstone impacted in the cystic duct, with hydrops of the gall bladder, and another stone impacted in the common duct, with obstruction to the biliary flow. I should say parenthetically that there is no doubt that she had obstructive jaundice with complete obstruction. Of course, it is said that a palpable gall bladder is rare in the presence of common-duct stone. In other words, if this mass had been gall bladder, it would have ruled against the presence of stone. One other bit of evidence is that the mass is said to have been fixed on respiration. Ordinarily, I think

the gall bladder would tend to move on respiration. If it was gall bladder and if it was fixed, can that be taken as evidence of tumor of the gall bladder that has extended to surrounding structures and thus caused fixation? The only other evidence that I can glean from the record is the tendency to leukopenia—a rather curious blood formula for a leukopenia in that the polymorphonuclear cells were at a normal or slightly high level, the lymphocytes were decreased, and the monocytes were increased. This is not the ordinary type of leukopenia. Whether one can lean very much on that observation, I do not know. In cirrhosis of the liver, a leukopenia is common, and this might be evidence that the patient had an underlying cirrhosis. The mass felt might have been an abnormal lobe of the liver, and there might have been a tumor of the liver on the basis of underlying cirrhosis. That seems to me to be arguing quite far afield on slim evidence.

There is no mention of a palpable spleen, and in cases of cirrhosis of the liver without splenomegaly not much leukopenia is found. Occasionally, this blood formula is seen in lymphoma, more commonly with a slight increase in lymphocytes, rather than a decrease. I think that again is very slim evidence. In the patient whom I saw a year ago, the white-cell count as I remember it was between 9000 and 10,000.

I shall mention one more thing regarding the leukopenia. If there had been an impacted stone in the cystic duct, the chances are that there would tend to be some fever, presumably dependent on rather low-grade inflammation, and we might expect to have at least a slightly elevated white-cell count and slight fever.

One always ought to bear in mind the possibility at any age of toxic hepatitis, so-called "catarrhal jaundice." I remember having had a patient operated on for obstructive jaundice due to stone, I thought, who proved to have nothing but benign catarrhal jaundice. The mass that was felt in the case under discussion rather rules out this diagnosis. One would expect to feel a tender liver in a case of jaundice caused by toxic hepatitis. This mass is not spoken of as being very tender. I mention this because one should always think of it, but I cannot see any really good reason why this patient should not have been operated on for the purpose of establishing a definite diagnosis and also with the hope of being able to re-establish some form of biliary drainage, and to rule out the possibility that a stone caused this condition. With the setup as we have it, I come back to my original diagnosis: that this patient had a chronic cholecystitis and cholelithiasis and that as a result, as sometimes happens, she developed a carcinoma of the gall bladder that extended to involve the biliary tract.

DR. JACOB LERMAN: Are you disturbed at all by the fact that the mass was smooth, if the examiner was accurate in his description?

DR. RICHARDSON: No, I do not believe I am. I am no more disturbed by his calling it smooth than I would be impressed by his calling it irregular.

DR. WILLIAM B. BREED: Nor by the fact that it did not move on respiration!

DR. RICHARDSON: That is a little more definite.

DR. BREED: Yes, but mistakes are made on that point just as often as on the other.

CLINICAL DIAGNOSIS

Carcinoma of head of pancreas.

DR. RICHARDSON'S DIAGNOSES

Chronic cholecystitis.
Cholelithiasis.
Carcinoma of gall bladder.

ANATOMICAL DIAGNOSES

Carcinoma of pancreas.
Cholelithiasis.
Hydrops of gall bladder.

PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: This patient was explored, and a huge distended gall bladder was found in which were many small stones and a large amount of colorless bile. The exploration was continued, and a firm mass was palpated in the pancreas, which was biopsied and showed carcinoma. The gall bladder itself did not show cancer. There was then a distinct problem in the surgical relief of this patient. Originally, it was believed that cholecystgastrostomy might be done, but with white bile in the gall bladder, it seemed probable that the cystic duct was obstructed and that such an operation would therefore be ineffective. Fortunately, the common duct was markedly dilated, and it was possible to anastomose the common duct successfully to the duodenum. The gall bladder was emptied of its stones and likewise drained. The patient made a fairly comfortable postoperative recovery, and the jaundice, though still present, was steadily diminishing at the time she left the hospital.

A PHYSICIAN: Was x-ray treatment given?

DR. MALLORY: I do not know. There is no mention of x-ray treatments in the record. If they are being given, I am sure it is with no enthusiasm.

CASE 28242

Presentation of Case

A sixty-five-year-old Russian tailor was admitted to the hospital because of loss of weight and appetite.

The patient was in good health, except for occasional bouts of sinusitis, until about six weeks before entry, when there was gradual onset of weakness and anorexia. Constipation appeared, requiring enemas every third day. The stools were dark, but not bloody. There was no distention of the abdomen and no pain. Seven pounds in weight was lost. Ten days before entry, a local physician diagnosed "anemia," and prescribed iron and vitamin pills. No improvement followed this medication. In the week before entry, some cramping was noted in the abdomen.

The family history was irrelevant. Four years before entry, the patient had a large thrombosed hemorrhoid excised in the emergency ward of the hospital. He had also been treated for sinusitis, on two occasions, in another department of the hospital.

On admission, the patient appeared well developed, but rather undernourished. The heart was normal. The right lung field was clear, but many moist inspiratory rales were heard at the left base. The abdomen appeared full. No free fluid or masses were palpable. There was some hypogastric tenderness, but no spasm. Peristalsis was hyperactive, with high-pitched sounds. The prostate was of normal size.

The temperature was 99.5°F., the pulse 80, and the respirations 20. The blood pressure was 100 systolic, 55 diastolic.

Examination of the blood showed a white-cell count of 21,700 with 78 per cent polymorphonuclears, 9 per cent large lymphocytes, 5 per cent monocytes and no abnormal cells. The red-cell count was 4,010,000. The serum protein was 4.5 gm. per 100 cc. The chlorides were 102.5 milliequiv. per liter, and the nonprotein nitrogen 39 mg. per 100 cc.

A roentgenogram of the large bowel after a barium enema showed no evidence of obstruction. The cecum was high, and the sigmoid was rather redundant. Two diverticula projected from the descending colon. On re-examination, numerous dilated loops of small bowel were noted close to the cecum, suggesting a lesion at the ileocecal valve. Roentgenograms of the upper gastrointestinal tract showed some deformity of the duodenal cap suggestive of old duodenal ulcer. There was definite obstruction to the passage of barium by an irregular, apparently ulcerated mass in the upper jejunum. The loops of small bowel below this level appeared filled with air.

A Miller–Abbott tube was passed, and a blood transfusion was given. The temperature rose to 101.5°F., and then dropped to normal. On the third hospital day, an operation was performed.

Differential Diagnosis

Dr. E. Parker Hayden: The history, at its outset, with a loss of weight and appetite of six weeks' duration, certainly suggests cancer. I shall exclude the possibility of cancer of the stomach simply on the failure of the X-ray Department to state that the stomach itself showed any abnormality when examined.

The onset of constipation six weeks before entry may or may not mean anything. Perhaps the patient was eating lightly or had shifted to a low-residue diet. The stools were not bloody. There was no distention of the abdomen and no pain. The serum protein was low, but the chlorides were normal. The nonprotein nitrogen was moderately elevated. The findings in the left chest may or may not be of much significance in an elderly person. The onset of definite cramps and hyperactive peristalsis indicates that the patient had mechanical obstruction on admission to the hospital, but on the other hand, the presence of some fever indicates infection — which is primary and which secondary, we must decide. The x-ray report states that there were dilated loops of small bowel close to the cecum. Whether these happened to lie close to the cecum, or actually involved the terminal ileum, is not certain. The diverticula in the descending colon, I suspect, were merely coincidental and, as so often happens, were probably not concerned in this picture.

We have evidence of an ulcerative mass in the jejunum and also evidence of obstruction near the ileocecal valve. The problem is to decide whether these findings were due to one lesion, perhaps primary at the ileocecal valve with adherence and involvement of a loop of jejunum, or whether there were two independent lesions. If there were two independent lesions, the condition might have been regional ileitis, or there might have been two independent cancers. It is difficult to be sure. Anemia and loss of weight are very common early symptoms of carcinoma of the cecum before localizing symptoms develop, and the subsequent onset of obstruction could readily have been due to involvement of the ileocecal valve with tumor. That diagnosis seems probable. The mass involving the jejunum could have been primary, or involvement secondary to a tumor in the ileocecal region. The localized loops in the region of the cecum also suggest the possibility of a herniation of small bowel through some aperture in the region of the cecum, a condition met with occasionally. However, I think I shall make a diag-

nosis of carcinoma of the cecum, at the ileocecal valve, with secondary involvement of the jejunum.

DR. RICHARD SCHATZKI: I do not know whether the record states emphatically enough that the films show no evidence of disease in the colon. The cecum is well outlined. There are several dilated loops of small intestine in the lower abdomen.

DR. HAYDEN: I was making some allowance for the fact that the roentgenogram does not always reveal lesions in the cecum.

DR. SCHATZKI: I think the fluoroscopy note, together with the films, is enough evidence against a lesion in the cecum. The upper gastrointestinal tract shows this ulcerated lesion in the first or second loop of jejunum, which has the definite appearance of malignant tumor. Some moderately but, I should say, definitely air-filled loops of small intestine are visible below this region, indicating additional involvement of the bowel below the region of ulceration. I do not see any films showing the small intestines filled with barium lower down, not because the small intestine was obstructed but apparently because such films are not here or they were not taken.

DR. HAYDEN: The loops described in the report, then, happened to be lying close to but were not concerned with the ileocecal valve.

DR. SCHATZKI: Not so far as there is evidence in the film. If there was a lesion there, it was in the small intestine.

CLINICAL DIAGNOSES

Intestinal obstruction.
? Lymphoma of intestine.

DR. HAYDEN'S DIAGNOSIS

Carcinoma of ileocecal region, with secondary involvement of jejunum.

ANATOMICAL DIAGNOSIS

Lymphosarcoma, multiple, of jejunum and ileum.

PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: At operation, it was found that this patient had multiple tumors of the small bowel. Each of these was several centimeters in length, and in each one the wall of the bowel was uniformly thickened all the way around its circumference. There was ulceration of the mucosa. The tumor masses were extremely friable, and in the course of exploration one of them spontaneously ruptured. It therefore became necessary to do a fairly extensive resection, and we eventually received three separate segments of bowel, which contained several tumors. On microscopic examination, all showed a rather undifferentiated type of lymphosarcoma. The patient died two days after operation.

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IMMEDIATE NEED FOR MEDICAL OFFICERS

All who were present at the annual banquet of the Massachusetts Medical Society and who listened to the speeches of Dr. Frank H. Lahey and Dr. Morris Fishbein appreciate the immediate need for medical officers in the Army of the United States. This crisis was referred to also by Dr. John F. Fulton in the Shattuck Lecture, when he stated that, by the end of this year, the air forces of the Army and Navy will be expanded to include about 20,000 flight surgeons and medical officers. In other words, NOW is the time to volunteer — not six months from now, or even a month from now. Furthermore, if a sufficient number of medical officers are not obtained by this means, there is every indication that more drastic measures will be taken to secure them.

The response in Massachusetts has been disappointingly slow, owing in a large measure to the erroneous impression of some physicians that the enrollment forms of the Procurement and Assignment Service are equivalent to applications for commission and to the fact that the information contained on these forms necessarily requires a considerable period before it can be tabulated and, hence, become available for use by the recruiting personnel of the armed forces.

To facilitate matters, as outlined in an editorial in the May 14 issue of the *Journal*, recruiting boards for medical officers have been established in all states. The office of the Massachusetts Medical Officers' Recruiting Board is located at 319 Longwood Avenue, Boston (ASP 3638). These boards, working in conjunction with the Procurement and Assignment Service, are authorized to commission directly a qualified physician in the Medical Corps of the Army, provided he is declared "available" by the state or local officers of the Procurement and Assignment Service; in such a case, the physician will be assigned to active duty within a few weeks following his application. In addition, the boards will forward applications for commissions from graduates of unapproved and foreign medical schools to the Office of the Surgeon General for individual consideration.

To repeat, the enrollment forms of the Procurement and Assignment Service are NOT applications for commissions. Furthermore, this governmental agency will NOT tell physicians what to do — whether to go, or when to go; its function consists only of acting in an advisory capacity to the armed forces and of determining whether physicians are "available" or "essential." This IS the call, and all qualified physicians in Massachusetts should IMMEDIATELY apply, through the Medical Officers' Recruiting Board, for a commission. After the war is over, let it be said that Massachusetts physicians VOLUNTEERED their services.

INDUSTRIAL HYGIENE AND THE WAR

THE recent joint meeting in Washington, D. C., of the National Conference of Governmental Industrial Hygienists and of the Subcommittee of Industrial Health and Medicine, Health and Medical Committee, Office of Defense Health and Welfare Activities, brought out many problems that are of vital interest in the present emergency.

The five major points raised by various speakers were as follows: workers, individually and collectively, should serve as the fulcrum of all efforts to improve industrial health; the efficacy of medical, engineering and chemical controls of occupational hazards has been proved; knowledge, experience and organization are such that time-loss due to disability can be greatly reduced; threats to the health of war workers have increased, in many parts of the country, to a critical, or even desperate, stage; and to meet this emergency,—that is, to avert disaster and to establish a condition of vigorous health among all workers,—all the facilities for prevention and restoration must be expanded.

Figures released by the War Production Board showed that seven and a half million people are now employed in producing the implements of war and that double that number will be needed by the end of the year. This marked increase will intensify existing problems and will create new ones. For example, the need for medical and nursing services in small plants, which now employ about two thirds of the industrial population and which, as a rule, fail to provide such services, will be even more acute. Furthermore, the employment of women will present difficulties that require a new approach, as will the introduction of new toxic substances, and longer working hours with shorter periods of relaxation.

State industrial-health officers praised the aid furnished by the Government—both financial and otherwise—and affirmed that they had the knowledge and the spirit markedly to decrease loss of time due to disability; however, they acknowledge that they were tremendously handicapped by a lack of personnel—physicians, engineers, chemists and nurses.

Since the outcome of the war depends on the production of its instruments, all agencies—federal, state and industrial—must co-operate in every possible way to increase productive efficiency by improving the health of war workers. And, after the battle has been won, this service, which is the fundamental right of the millions of industrial workers in this country, should be continued.

MEDICAL EPONYM

PFEIFFER BACILLUS

"Vorläufige Mittheilungen über die Erreger der Influenza [Preliminary Notes on the Etiologic Agents of Influenza]" was published by Richard Friedrich Johann Pfeiffer (1858), while he was a *Privatdozent* in the Institute for Infectious Diseases at Berlin, in the *Deutsche medicinische Wochenschrift* (**18**:28, 1892). A portion of the translation follows:

> In all the cases [31] of influenza, a distinct variety of bacillus was found in the characteristic purulent bronchial secretion. These rods were demonstrable in absolutely pure culture and usually in tremendous numbers in the cases of uncomplicated influenza. . . . The rods were found only in influenza. . . . The numbers of the bacilli kept pace with the course of the illness. . . . Numerous inoculation experiments were attempted. Positive results can be obtained only in monkeys and rabbits.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

CASE HISTORY: TOXEMIA FOLLOWED BY ANURIA AND DEATH

A twenty-eight-year-old primipara, when approximately thirty-five weeks pregnant, entered the hospital with marked edema, a blood pressure of 164 systolic, 92 diastolic, a trace of albumin and a sediment containing many granular and hyaline casts. She had been followed by her physician from the beginning of pregnancy and had shown no abnormalities until about the thirtieth week, when albuminuria appeared. The blood pressure was said not to have risen for another month. The past history was irrelevant; there had been no contagious diseases or any operation. The patient was treated at home for a week before she entered the hospital. Two days after admission, because of a sustained systolic blood pressure of 160 to 170

and a diminution in the urinary output, labor was induced with a Voorhees bag. The bag was expelled twenty-four hours later, and an infant, presenting by the breech, extracted. The baby weighed 5 pounds, 12 ounces, and lived only a few hours. Anuria developed immediately after delivery, and the patient died forty-eight hours later.

Comment. Although the care of this patient was adequate, it was not of the most intelligent nature. If she had been hospitalized at the onset of her symptoms, and if rest in bed and a diet of 2½ quarts of milk a day (with absolute restriction of salt) had been instituted, improvement might have been noticed. If, under hospital care and constant surveillance, edema had increased, the blood pressure had remained stable or increased, and the urinary output had decreased, delivery might well have been considered earlier. The use of the Voorhees bag for induction warrants no criticism. It is barely possible that rupture of the membranes alone would have achieved the same end, and that if an abrupt rise in the blood pressure had occurred during the hospital stay, an immediate cesarean section might have saved the baby and lessened the degree of nephritis that resulted in anuria and death.

Some cases of anuria developing in association with hypertension and toxemia, particularly those in which separation of the placenta (which this patient did not have) occurs, have been successfully treated by decapsulation of the kidney, but this treatment is not so satisfactory when applied to this type of anuria as it is for that following incompatible blood transfusion.

DEATH

POPOFF — Constantine Popoff, M.D., of Haverhill, died May 30. He was in his sixtieth year.

Born in Bulgaria, Dr. Popoff received his degree from Harvard Medical School in 1910. He was a lieutenant colonel in the United States Army Medical Reserve, and at the time of his death was awaiting orders to active service. He was a member of the New England Roentgen Ray Society and the Radiological Society of North America, and a fellow of the Massachusetts Medical Society and the American Medical Association.

He is survived by two daughters.

WAR ACTIVITIES

CIVILIAN DEFENSE

Blood and Plasma Banks

Regulations for the administration of the Blood and Plasma Bank Program of the Medical Division of the United States Office of Civilian Defense have now been prescribed, and funds are available for grants to assist approved hospitals in establishing blood and plasma banks. Only hospitals within three hundred miles of the Atlantic, Pacific and Gulf coasts are eligible for such grants. After July 1, 1942, these geographical restrictions may be modified, so that grants may be made to inland hospitals. Applications should be addressed to the Chief Medical Officer, United States Office of Civilian Defense, Washington, D. C.

Technical manuals on blood and plasma banks, prepared by the Subcommittee on Blood Substitutes of the Division of Medical Sciences, National Research Council, are now available for distribution on request of any hospital to the Chief Medical Officer, Office of Civilian Defense.

The Red Cross has established eighteen donor centers in various parts of the country, which are successful in obtaining an adequate supply of blood donors for military purposes. Blood for the production of dried plasma for civilian-defense purposes will also be obtained from these sources.

Hospitals that establish their own blood and plasma banks with the financial assistance of the Office of Civilian Defense are advised to build up their reserves of blood and plasma by expanding blood collections from relatives and friends of patients who are to receive transfusions. A public campaign for volunteer donors, which might compete with the work of the Red Cross, should be avoided if possible. If public solicitation is necessary, hospitals should appeal to the local chapters of the American Red Cross for assistance in recruiting hospital donors. Blood-donor campaigns by agencies other than the Red Cross will tend to confuse the public and may interfere with the blood collection by the Red Cross for the armed forces.

The official release, dated April 17, 1942, of the Federal Security Agency and the United States Public Health Service is as follows:

> Whereas, on April 11, 1942, there was allotted from the "Emergency Fund for the President" to the United States Public Health Service the amount of $292,500, "to be expended by said Public Health Service in connection with emergencies affecting the national security and defense for procuring and establishing either independently or, subject to regulations to be promulgated by the Surgeon General, by grants to public and private hospitals located not more than three hundred miles from ocean or Gulf Coast, reserves of liquid, frozen or dry blood plasma or serum albumin for the treatment of casualties resulting from enemy action," the following regulations are promulgated to govern the administration of this allotment:
>
> Section I. Eligibility for Grants
>
> Preference shall be given to hospitals serving communities whose geographical location implies a likelihood of civilian casualties from enemy action, and which are inadequately equipped to handle such casualties.
>
> To be eligible for a grant a public or private hospital located not more than three hundred miles from ocean or Gulf Coast (the Appropriation Act for the year 1943 may not limit grants to hospitals within this geographical area) shall:
>
> (1) Have a capacity of not less than 200 beds, exclusive of bassinets, provided that two or more smaller hospitals totaling 200 beds may submit a co-operative project designating one of the participating hospitals as the grantee;
>
> (2) Be on the approved list of the American College of Surgeons and the Hospital Register of the American Medical Association; and
>
> (3) Have on the professional staff a physician whose qualifications are the equivalent of those

required by the American Board of Pathology for its diplomates.

Section II. Approval of Plans

A grant shall cover a period of not more than twelve months following the approval of the plan, or not beyond June 30, 1943, and may be used only for the purchase of equipment necessary for the preparation of liquid or frozen plasma, reconditioning or minor alterations of existing quarters, necessary travel and subsistence allowance of $6.00 per diem to cover a training period, if required, of not more than one week, for the physician directing the blood-plasma project, and temporary salaries of personnel necessary for the establishment of a blood and plasma project.

The maximum grant for one hospital is $2000.

A hospital desiring to receive a grant shall submit a plan to the Chief Medical Officer, United States Office of Civilian Defense, who is authorized to receive such plans on behalf of the Surgeon General of the United States Public Health Service. A plan shall contain the following information:

(1) The number of hospital beds classified according to use;

(2) The name and qualifications of the physician who will direct the plasma project;

(3) Description of present blood and plasma project, if any;

(4) The type and amount of plasma reserves which the institution desires to prepare;

(5) The delivered price of equipment necessary to complete the existing facilities for preparing such plasma — such items to be numbered and described in accordance with the equipment inventory in *A Manual on Citrated Normal Human Blood Plasma*, issued by the Office of Civilian Defense, or equivalent approved substitute equipment.

(6) The materials or labor, if any, needed for adapting existing quarters to the needs of the blood plasma project; and

(7) The salaries, if any, to be paid additional personnel until the plasma reserve has been prepared. Salary items shall also show the proposed periods of employment for each individual and the proposed monthly rates of pay.

When a plan is recommended by the Chief Medical Officer of the Office of Civilian Defense for the approval of the Surgeon General, the hospital will be furnished a budget and acceptance form to be signed, notarized and returned to the Chief Medical Officer, Office of Civilian Defense.

Section III. Conditions of Grants

The hospital shall agree to build up a plasma reserve of at least one unit per bed within three months after delivery of the necessary equipment. A unit of plasma is that amount derived from 500 cc. of citrated whole blood, consisting of about 250 cc. of liquid plasma.

The agreed amount of plasma reserve shall be maintained for use without charge and only for treatment of casualties caused by enemy action. The reserve shall be released for use in other local hospitals for this purpose on order of the local chief of Emergency Medical Service and for transfer within the state on order of the state chief of Emergency Medical Service, or transfer from one state to another on the order of the regional medical officer, Office of Civilian Defense.

Liquid plasma shall be kept from being outdated by replacement of older by newer plasma. Replaced units may be utilized for current needs of the hospital in the treatment of its regular patients, provided the plasma reserve shall not be allowed to fall below the stated minimum.

All plasma shall be prepared in accordance with manuals of the Office of Civilian Defense prepared by the Subcommittee on Blood Substitutes of the National Research Council.

The hospital shall agree to continue the plasma project for its current needs after the expiration of the federal grant and to maintain for the duration of the war the minimum stated reserve; thereafter the reserve may be used by the hospital without restriction.

A record shall be kept of all blood donors, including their blood types, to expedite obtaining donors for emergencies.

No funds made available under the grant shall be used for the payment of blood donors.

Any blood plasma project under this program shall be subject to inspection by authorized representatives of the Surgeon General of the United States Public Health Service.

Section IV. Method of Payment

Payments will be made on a reimbursement basis for expenditures made in accordance with the approved budget. Applications for reimbursement shall be notarized and addressed to the Chief Medical Officer, United States Office of Civilian Defense. The procedure for payment will be as follows:

(1) Payments from the allotment to cover the purchases of nonexpendable equipment aggregating $300 or more will be paid upon receipt, from the authorized administrative head and accounting officer of the hospital, of an itemized statement of the purchases supported by invoices showing the date of delivery of such equipment;

(2) Payments will be made for the authorized training expenses of the physician who is to direct the blood plasma project whenever the hospital presents a notarized claim itemizing the travel and per-diem allowance incident to the training;

(3) Reimbursement for other items of the approved budget will begin only after actual production of blood plasma is started. During the first three months of production, reimbursement will be made on a monthly basis and quarterly thereafter for the duration of the grant. Such reimbursement will be made only upon receipt of a report form prescribed by the Surgeon General from the institution showing expenditures incurred during the period, total plasma prepared during the month, and the total reserve on hand to date;

(4) Payments may be withheld, and plasma produced as part of this project may be transferred by the Surgeon General, from any hospital which fails to meet the conditions of the grant or to comply with the regulations:

(5) Each hospital shall submit monthly reports during the period of the grant showing the amounts of plasma on hand and used — thereafter, for the duration of the war, the hospital shall submit such reports quarterly on its use of the plasma; and

(6) Hospitals shall submit promptly reports including clinical abstracts of any untoward experiences encountered in the use of plasma for the duration of the war.

MISCELLANY

NOTES

The Committee on Faculty at Middlesex University has announced the appointment of Dr. Ernst Mathias to the professorship of pathology in the School of Medicine, to fill the vacancy caused by the recent death of Dr. Leon K. Baldauf. Dr. Mathias was born in Koenigsberg, Germany, and studied at the Universities of Heidelberg and Koenigsberg, receiving his M.D. degree from the latter institution in 1912. He served as a member of the Department of Pathology at the University of Koenigsberg and at the University of Breslau, and was decorated three times for his services in the medical corps during World War I. Returning to teaching, he became professor of pathology in the medical school of the University of Breslau, and pathologist of the Jewish Hospital in that city. He visited the United States first in 1937, when he received an honorary fellowship in pathology from Yale University. He was forced to leave Germany by the Hitler regime, came to this country permanently in 1938, and received an appointment as pathologist in hospitals at Northampton, Holyoke and Greenfield. Dr. Mathias is a member of the American Association of Pathologists and Bacteriologists and of the New England Pathological Society.

Middlesex University graduated a class of eighty-six from the School of Medicine at commencement exercises held on June 1 in Waltham. Congressman Thomas H. Eliot delivered the commencement oration. Dr. Stephen Rushmore, dean of the school, presented the candidates, and the medical degrees were conferred by President C. Ruggles Smith.

At the annual meeting of the New England Society of Physical Medicine, held at the Ring Sanatorium and Hospital on Wednesday, June 3, the following officers were elected: Dr. Arthur L. Watkins, of Boston, president; Dr. Wilmot L. Marden, of Lynn, first vice-president; Dr. David C. Ditmore, of Boston, second vice-president; Dr. William D. McFee, of Boston, secretary; Dr. Howard Moore, of Boston, treasurer; and Drs. Charles W. McClure and Frank B. Colloten, of Boston, councilors for three years.

REPORTS OF MEETINGS

EVANS MEMORIAL HOSPITAL

At an Evans Lecture at the Evans Memorial Hospital on January 23, Dr. Alfred Blalock, of Baltimore, spoke on "The Pathogenesis and Physiology of Traumatic Shock."

Shock is a manifestation of vascular failure, which may be cardiac or peripheral vascular in origin. Syncope of cardiac origin, as exemplified by the Stokes–Adams syndrome or any reflex slowing, such as that caused by a sensitive carotid sinus, is characterized by bradycardia and fainting. Its onset is sudden, and its duration is short, as contrasted with cardiac collapse. The cause is myocardial trauma of any origin, whether from coronary-artery disease, diphtheria or tamponade. There is abrupt tachycardia. In cardiac shock, there is usually congestion of some part of the vascular tree, either peripheral or pulmonary, in contradistinction to peripheral vascular shock. Both types may have coldness, sweating and palpitation, and an acute cardiac shock may be difficult to distinguish from peripheral vascular shock.

By definition, shock is a condition in which peripheral vascular failure is brought on by a discrepancy between the existing vascular bed and the circulating blood volume, which leads to tissue anoxia. Shock may result from an increase of the vascular bed or a decreased circulating volume or a combination of both. The last is probably the commonest kind. The characteristics of early shock, which is the optimum time for treatment, are hemoconcentration, rapid pulse and low blood pressure. The last manifestation is late and is not comparable with the others for early recognition. However, a chart of the blood pressure over a period is the best way of evaluating the status of an established case of shock. Of 2 patients with equal blood-pressure readings, the one with warm extremities who has not yet called on his vasoconstrictor apparatus has a better prognosis than the one with cold extremities whose sympathicoadrenal system has already reacted maximally.

The remainder of the discussion was concerned with secondary or hematogenic shock, in which there are vasoconstriction and an increased vascular bed. Studies reveal that immediate gross hemorrhage causing death of an animal results in none of the tissue changes considered characteristic of shock, for there is apparently no time for its development. Slow bleeding, however, produces changes similar to those of shock, as evidenced by the fact that transfusion of more than the lost volume of blood after a prolonged period of lowered blood volume and blood pressure will not save the animal. The tissues become hyperemic and necrotic. Dunphy and his associates have shown by early sacrifice of animals after trauma or burns that there is no capillary damage, much loss of fluid or tissue necrosis. But shock from all causes has identical terminal tissue lesions, so that it is only by study of early cases that any differentiation can be made.

Shock may be caused by many things, but the most important are toxemia, local fluid and blood loss, and nervous trauma. The reason for most of the controversy is a failure to recognize that this syndrome cannot be explained by one mechanism. Experimentally, furthermore, there is a discrepancy in terminology, method of producing shock, anesthetic used, species difference and criteria of shock. Dr. Blalock employs the method of traumatizing one lower extremity but amputates higher than formerly — that is, at the midabdomen. In such animals, there is a local loss of fluid at or near the site of trauma amounting to half the body volume, and this is the cause of shock. The seriousness of this local fluid loss is also borne out by longer experiments on bowel handling, which allows for possible accumulation of toxic products. Here, the loss is plasma rather than whole blood. In burns, 3 per cent of the body weight may be lost locally, and a similar loss in normal dogs is sufficient to cause death in twenty-five hours. This may not be the only cause, but it is the most important initial one in early deaths from burns, which are most numerous. Other factors, such as sepsis, enter into later deaths. And since burns are causing a large percentage of deaths in the present war, the necessity of finding a satisfactory method of preventing excess local fluid loss is evident. Some form of tanning is advisable for flat surfaces.

Experimental work is being carried out on the so-called "crush syndrome," but so far this has been of little practical value. However, such injuries are not rare in people

pinned for long periods under debris of bombed buildings. The main differences between the experimental animals and crushed people are the immediate changes in the former on being released in contrast with the later changes in man. In animals, those treated by pressure to the part survived in 14 out of 17 cases, whereas the control group had only 1 survival in a similar number. This was in spite of an elevated hematocrit reading and fluid loss. Therefore, treatment should consist of constant pressure to the part, with an elastoplast bandage or tourniquet to prevent excessive fluid loss in the part. Blast injuries are known to be fatal in certain cases in which no evidence of any direct trauma is present. About 40 to 50 per cent of fatalities from this cause show no signs of direct violence (Hatfield). Such persons reveal widespread pulmonary hemorrhages but not of such magnitude as to cause death by compression. In animals subjected to such forces, similar bilateral pulmonary hemorrhages are found. A blast may cause similar injuries to the brain if the head is movable, and may result in intracellular neuronal disturbances. However, such cerebral damage is considered to be an exaggerated feature by some observers.

Acute circulatory failure may be classified as hematogenic (hemorrhagic), neurogenic (spinal) or vasogenic (histamine). In the last two, the cardiac output decreases before the blood pressure, whereas in the vasoconstriction following hemorrhage and trauma to muscle, the blood pressure drops first. The degree of hemoconcentration is proportional to the loss of blood or plasma.

In the treatment of shock, adrenocortical extract is theoretically useful but does not seem to have lived up to expectations practically. A pure oxygen intake, although likewise based on good logic, has not proved practically valuable. The application of heat is advisable if the person is in the cold or has just been brought in, but the great value of heaters and blankets in the usual case seems overemphasized. Experimentally, there have been more and longer survivals of animals left relatively cold. Furthermore, there has been this same improvement in results with the local application of cold, despite the same degree of fluid loss. Granted that the amount of heat supplied was physiologically excessive, it does seem illogical to use heat, which draws blood to the part from more vital centers. When the circulating volume is therapeutically increased, the addition of heat is apparently not contraindicated. Regarding parenteral fluid administration, it was emphasized that large amounts of crystalloids not only are lost but also wash out with them large amounts of plasma protein, thus decreasing the osmotic pressure, and are, therefore, definitely deleterious. Transfusions are dangerous if incompatibilities exist, and this is another indication for the use of plasma or serum. Patients die of loss of blood volume rather than of erythrocytes.

In dangerous degrees of shock, hemoconcentration may sometimes not be found. The pulse rate is often of little value, whereas the blood pressure is a fair but late indication of shock. It is still the best gauge of response to therapy. A clinical evaluation of the situation based on the severity of the injury and the amount of blood loss is often more valuable than the blood pressure. In other words, one should not wait for changes in pulse, blood pressure or hemoconcentration before initiating shock treatment. One should never administer more than 1500 cc. of saline solution daily.

Thus, it is seen that the mechanism of certain types of shock is understood, such as the local loss of tissue fluids, but that there are many others. In general, an attempt should be made to replace the same kind of fluid as that lost. The use of whole blood is contraindicated only when hemoconcentration is a prominent feature, as in burns. Plasma and serum are available and very useful. Cohn's albumin appears to be a hopeful blood substitute. This is a nonantigenic, hypertonic solution.

WILLIAM HARVEY MEDICAL SOCIETY

At a regular meeting of the William Harvey Medical Society of Tufts College Medical School at the Beth Israel Hospital on January 30, Dr. Chester S. Keefer spoke on "The Epidemiology of Hemolytic Streptococcus Infections." The early recognition that a single organism was responsible for the many diseases attributed to the hemolytic streptococcus was made difficult by the multitude of clinical manifestations, but it is now generally accepted that this is a single organism.

Hemolytic streptococcus infections account for 5 to 10 per cent of all sickness and cause about 70 per cent of infections in wartime, burns and the puerperium. In crowded communities, such as war camps, it is surpassed only by measles and epidemic influenza. It is the commonest cause of bacteremia, as well as the commonest initiating factor in acute glomerulonephritis and in recurrences of rheumatic fever. Fortunately, there has been a great improvement in treatment in the past few years with the advent of the sulfonamides.

Some advances have been made in the epidemiology of hemolytic streptococcus infections, but knowledge is still far from complete. This is especially true of the control of its spread, and methods at present are inadequate. Predisposing factors include age and season of the year. Between five and fifteen years is the commonest period for most streptococcal infections attributable to the throat, whereas puerperal fever is commonest from twenty to forty years of age. The incidence of erysipelas, on the other hand, is greatest under ten and over forty years of age. The seasonal prevalence in New England — the winter months from December to March — parallels the incidence of respiratory infections. Any wound is likely to develop some degree of infection if the normal skin barriers are interrupted. This is particularly true of burns and war wounds. Mortality rates are highest under four years of age.

The modes of spread are many. The normal habitat is largely in the throat and nose. Carriers are commonest in the winter. Infection may be transmitted by contact, hands of patients, droplets through the air and dust, and fomites in the form of clothing, food and toys. Autoinfection is not rare. Five to thirty per cent of the population are carriers of the hemolytic streptococcus, but only about a third of these harbor the significant Group A organism. The carrier state is commoner in children than adults and varies with the season, crowding, contacts and the number of respiratory infections.

Classification, as carried out by blood-agar methods, has resulted in Groups A to H, whereas in Group A there are more than twenty-eight types determined by agglutination (Griffith) and precipitation (Lancefield). The soluble products of the hemolytic streptococcus are the erythrogenic toxin, hemolysin (streptolysin), leukocidin, fibrinolysin and a spreading factor. All have been shown to be antigenic, except leukocidin, which probably is, and the spreading factor, about which little is known. The cellular components are as follows: an M substance, which is type specific and is used for the production of antiserum; a T substance, which is separable by agglutination; a C substance, which is a group-specific carbohydrate; an N.P. substance, which is a nucleoprotein and is not even species specific; and hyaluronic acid, which forms part of the cap-

sule but is not antigenic. The experienced observer can usually tell the groups from blood-agar plates, but it is better to use precipitation methods with specific antiserums. The type-specific M substance of Group A organisms, unlike that of the pneumococcus and other organisms, is protean in character.

The most characteristic sources of Group A are human infections; of B, bovine mastitis; of C, most animal streptococcal diseases; and of D, cheese and the human body (enterococci, certain saprophytes, organisms occasionally encountered in endocarditis and Ludwig's angina). Some of the higher groups are found in pneumonia, sinusitis, agranulocytic angina, some normal human beings, milk and dogs.

There are certain well-defined phases of streptococcal infections: a toxic phase, lasting from three to seven days and causing scarlet fever and so forth; a septic phase, lasting three to seven days or longer and causing severe localized reaction and only occasional bacteremia, such as the septic sore throat; a latent phase, lasting from ten days to six weeks in which an apparently normal convalescence is marred by a recrudescence; and a late nonsuppurative phase, which may last from a few days to an indeterminate length of time. The last manifestation consists of fever, lymphadenopathy, nephritis, edema, cardiac insufficiency, arthritis, splenomegaly, with hepatomegaly, and erythema nodosum. At present, there is no adequate method for preventing or protecting against this phase of the infection. There may be only a persistent low-grade fever following a sore throat, and the origin may not be discovered except by the taking of an electrocardiogram. There may be edema and cardiac insufficiency without true acute glomerulonephritis, and cerebral edema, with hypertension, may occur. Convulsions may be controlled by magnesium sulfate. It is best to prevent this phase, if possible, because of the danger of permanent cerebral damage. The toxic phase may be very acute and severe, but may be controlled with large amounts of antitoxic serum. This form of therapy, however, may merely serve to relieve the toxicity temporarily while allowing septic manifestations, such as acute otitis media. In this way, the septic phase not infrequently merges into the toxic. The sulfonamides have decreased the bacteremic mortality from 75 to less than 20 per cent.

Over a period of two years in a large general hospital, 95 per cent of the streptococcal cases were caused by Group A, whereas Groups B and C were responsible for only rare human illnesses. Group B has been identified in cases of puerperal fever, endocarditis, pyelonephritis, diabetes, pneumonia, meningitis and arthritis. Of the cases studied, by far the largest number were scarlet fever (414), followed by tonsillitis (120), otitis media and mastoiditis (131), and then by a great variety of other diseases. Of the scarlet-fever cases, 75 per cent were caused by five types of streptococcus, which caused 53 per cent of the other miscellaneous infections. Types 15 to 17 caused 36 per cent in the former and 22 per cent in the latter group. This prevalence of types varies with the year, community and country, but in any one outbreak in a community there is one or a small number of streptococcal types. Therefore, tracing of cases by typing is not difficult if the number is small.

In a study of multiple cases of scarlet fever in families, the same type in a family was found in 100 per cent of 10 cases, and only six different types were found in all 36 cases. Therefore, spread undoubtedly occurred by actual contact. Examination of the types in streptococcal complications reveals that the same type as that of the original focus is frequently (in 11 out of 12 cases) found in less than two weeks, whereas it is infrequently so (in 3 out of 18) after two weeks. It may be concluded, then, that later complications are largely the result of cross infection in a ward or hospital, and this, therefore, becomes an important public-health problem. Typing methods may be used to advantage in tracing cases. Strict isolation and segregation of the same types in scarlet fever and other epidemics should be carried out if at all possible or practical.

At the present time, control of infection with the hemolytic streptococcus may be approached by the isolation of the patient, the tracing of the source, and the observance of strict surgical asepsis in patients with wounds, burns and puerperal infections. At least 70 per cent of wounds and burns develop such an infection in the first week, probably from other patients, attendants, clothes and dust. The highest bacterial counts in the air occur in the morning, when beds are made and the wards cleaned, and dressings should preferably be postponed to another time.

BOOK REVIEWS

Handbook of Communicable Diseases. By Franklin H. Top, M.D., M.P.H., and collaborators. 8°, cloth, 682 pp., with 73 illustrations and 10 color plates. St. Louis: The C. V. Mosby Company, 1941. $7.50.

This is a sound, up-to-date book on the diagnosis, treatment, general management and prophylaxis of all the communicable diseases, with particular attention to the public-health angles involved. Anyone who needs such a book, and who does not own one, will do well in buying this. The only question that arises is, In a field already so well supplied with adequate texts, was there any real need to add another to the list? Most authors along this line are in substantial agreement, and the reviewer cannot help believing that there is small profit in rehearsing, in more or less stereotyped fashion, matters already sufficiently well covered in most libraries.

The Proceedings of the Charaka Club. Volume X. 8°, cloth, 270 pp., with 9 plates and 34 figures. Baltimore: The Williams and Wilkins Company, 1941. $5.00.

This volume contains a selection of papers read before the Charaka Club during the years 1937 to 1940. Of particular interest is a paper on the early years of the club, by Dr. Bernard Sachs, one of the original members. A timely publication is that of Dr. Walter R. Steiner on the experiences of a physician in the United States Sanitary Commission during the Civil War. There are twenty-one other papers, mostly representing the literary interests of physicians.

Training and Efficiency. By E. Jokl, E. H. Cluver, C. Goedvolk and T. W. De Jongh. 4°, boards, 188 pp., with 28 illustrations and 18 plates. Johannesburg, South Africa: The South African Institute for Medical Research, 1941. 10s. 6d.

This book is a study, made by the South African Institute of Medical Research, of 32 substandard army recruits, presumably a fairly representative cross section of a large group of youth in that political division of the Continent. It was undertaken to determine if, by compulsory training, this class of the citizenry could not be raised to a level of efficiency that would permit of their being classified as useful members of society.

One of the results of this study was to show that this substandard condition is not the outcome of fundamental biologic defects but rather is due to environmental conditions. Primarily, the physical fitness of a large proportion of Europeans in South Africa was lamentably low, and very many of the subjects had not the initiative to overcome this condition.

It appears from the success of this experiment that there is very good reason to think that by means of compulsory physical training, supplemented by appropriate occupational instruction, the labor power of this section of society may be greatly increased and that the opinion, so often expressed, that this class is "utterly degenerated" may thus be completely refuted. As a sociologic study, the book deserves to be read, and its recommendations should be heeded.

The Therapeutics of Internal Diseases. Vol. IV and Vol. V. Supervising Editor: George Blumer, M.A. (Yale), M.D. Associate Editor: Albert J. Sullivan, M.D. 4°, cloth. Vol. IV, 790 pp., with 56 illustrations, 22 tables and 2 charts. Vol. V, 765 pp., with 29 illustrations, 31 tables and 1 chart. New York: D. Appleton-Century Company, Incorporated, 1941. Sold only as a set (5 vol. — $50.00).

These two volumes complete the set, and a general index is supplied to all five volumes. Each section has been written by a man particularly interested in the system of the body under discussion. Volume IV deals with the diseases of the gastrointestinal tract, the genitourinary organs, the kidneys, the blood and the locomotor system. Volume V is concerned with diseases of the nervous system, metabolism, endocrine glands and skin, with chapters on the vitamins, allergy and chemotherapy, with sulfonamide drugs. The authors are outstanding in their respective fields, and the volumes are thoroughly sound and up to date.

The whole set can be highly recommended, for it maintains a standard rarely equaled in books of this type. Great credit should be given to the expert editing of Dr. George Blumer and his associate, Dr. Albert J. Sullivan.

Biological Symposia: A series of volumes devoted to current symposia in the field of biology. Edited by Jaques Cattell, Ph.D. Vol. III. *Muscle.* Edited by Wallace O. Fenn, Ph.D. 8°, cloth, 370 pp., with 15 tables and 82 figures. Lancaster, Pennsylvania: The Jaques Cattell Press, 1941. $3.50.

The editor of this interesting volume points out that care has been taken to include an aspect of muscle physiology that has gone out of fashion in this country and has been almost totally neglected until recently. However, the rapid progress in this field in recent years justifies the hope that a reasonably complete understanding of the actual mechanism of muscle contraction is not beyond reach. The contributing authors and their topics are as follows: "Introduction to Muscle Physiology," by Wallace O. Fenn; "Muscle Function as Studied in Single Muscle Fibers," by Robert W. Ramsay and Sibyl F. Street; "Muscle and the Heart's Motto," by Frederick H. Pratt; "Muscle Excitability," by Henry A. Blair; "Action Potentials and Conduction of Excitation in Muscle," by Elil Bozler, with a discussion by A. S. Gilson, Jr.; "Conduction in Smooth Muscles," by Arturo Rosenblueth; "The Local Activity around the Skeletal Neuromuscular Junctions Produced by Nerve Impulses," by T. P. Feng; "Action Potentials of Skeletal Muscle," by Allan C. Young; "The Regulation of Energy Exchange in Contracting Muscle," by Dugald E. S. Brown; "The Action of Muscles in the Body," by Herbert Elftman; "Changes during Muscle Contraction as Related to the Crystalline Pattern," by Ernst Fischer, with a discussion by Francis O. Schmitt; "The Significance of Oxidations for Muscular Contraction," by Otto Meyerhof; "The Efferent Innervation of Muscle," by C. A. G. Wiersma; "On the Nature of Certain Diseases of the Voluntary Muscles," by G. D. Gammon, R. L. Masland and A. M. Harvey; "Theories of Electrolyte Equilibrium in Muscle," by Robert B. Dean; "Electric Potential Changes Accompanying Neuromuscular Transmission," by J. C. Eccles, B. Katz and S. W. Kuffler. All these chapters make interesting as well as provocative reading, and no doubt every library will wish to have a copy of this volume on its shelf for reference by students and investigators in this field.

Occupational Diseases: Diagnosis, medicolegal aspects and treatment. By Rutherford T. Johnstone, M.D. 8°, cloth, 558 pp., with 132 illustrations and 26 tables. Philadelphia: W. B. Saunders Company, 1941. $7.50.

This volume furnishes, in a compact form, information regarding occupational diseases. The author states that his fundamental aim is to outline a basis for the diagnosis and treatment of the commoner occupational diseases, to interpret the medical and legal phase, and to predict, from experience, the expected disability. A considerable amount of space is allocated to workmen's compensation laws in the various states and to the function of the physician as related to such compensations.

Chapters are devoted to each of the common diseases met with in industry. They are condensed to furnish valuable information, so that it is unnecessary to read a more voluminous treatise. A considerable number of case histories are given to illustrate the various diseases. The bibliography is extensive, and the book is well illustrated.

This book should be of particular value to the general practitioner.

The Art and Science of Nutrition: A textbook on the theory and application of nutrition. By Estelle E. Hawley, Ph.D.; and Grace Carden, B.S. 8°, cloth, 619 pp., with 140 illustrations. St. Louis: The C. V. Mosby Company, 1941. $3.50.

The problems of nutrition are always of first importance. And if, in wartime, they are not of enhanced significance, at least they are more difficult of practical solution. What with increased food costs, rationing and, perhaps, difficulties in distribution, nutritionists are likely to find it hard to maintain adequate standards.

Sound books on nutrition will therefore be — as they should — studied with particular care. And this is a sound book. It discusses the general nutritional requirements of the body; the various food elements, qualitatively and quantitively, that must go to maintain them; food requirements under special conditions; the diet therapy of various diseases; and lastly (in a section of more than a hundred pages), the choice, preparation and serving of foods.

It is, as most such books are, designed rather for reference than for continuous reading, and its value is enhanced by numerous charts, tables and recipes. Its weakest feature is its pictures, some of which are poorly reproduced and others seem to be of slight importance, if not actually superfluous.

(Notices on page viii)